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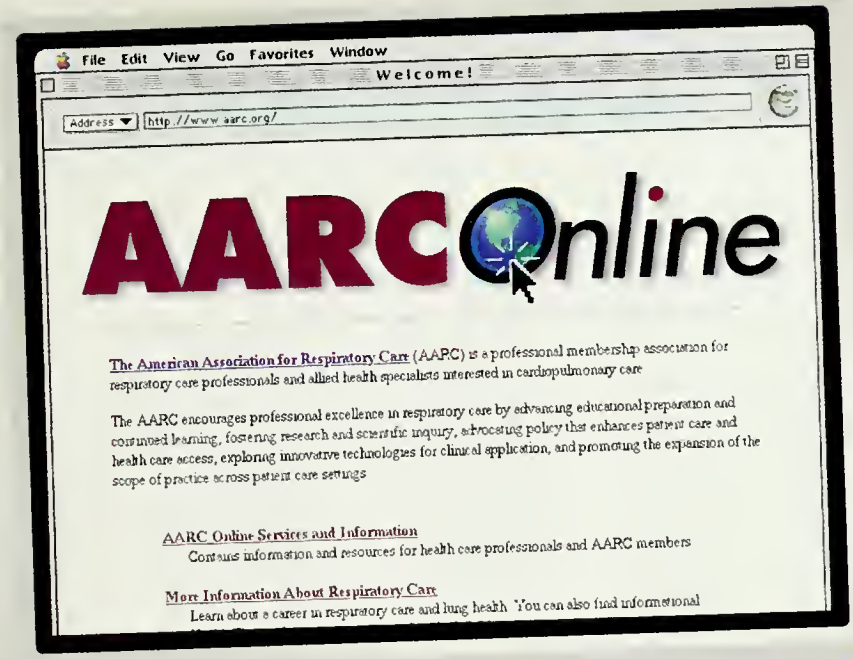
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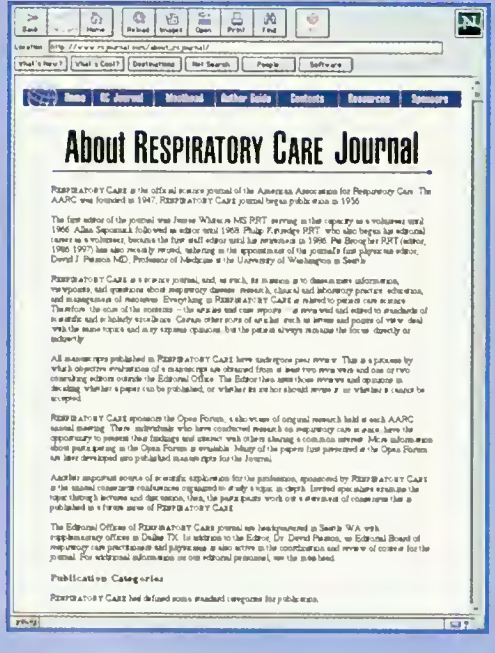

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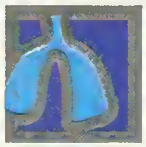

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Editorials, Commentaries, and Reviews To Note

Trends in Drug-Resistant Tuberculosis in the United States, 1993-1996—Moore M, Onorato IM, McCray E, Castro KG. JAMA 1997;278(10):833-837; **Long-Term Hospitalization for Tuberculosis Control: Experience with a Medical-Psychosocial Inpatient Unit**—Singleton L, Turner M, Haskal R, Etkind S, Tricarico, Nardell E. JAMA 1997;278(10):838-842; **Detention of Persistently Nonadherent Patients with Tuberculosis**—Oscherwitz T, Tulsky JP, Roger S, Sciortino S, Alpers A, Royce S, Lo B. JAMA 1997;278(10):843-846. **Defending the Public's Health against Tuberculosis (Editorial)**—Reichman LB. JAMA 1997;278(10):865-867.

Transmission of a Highly Drug-Resistant Strain (Strain W1) of *Mycobacterium Tuberculosis*: Community Outbreak and Nosocomial Transmission via a Contaminated Bronchoscope—Agerton T, Valway S, Gore B, Pozsik C, Plikaytis B, Woodley C, Onorato I. JAMA 1997;278(13):1073-1077; **Transmission of *Mycobacterium Tuberculosis* by a Fiberoptic Bronchoscope: Identification by DNA Fingerprinting (Brief Report)**—Michele TM, Cronin WA, Graham NM, Dwyer DM, Pope DS, Harrington S, et al. JAMA 1997;278(13):1093-1095; **Tuberculosis Infection after Bronchoscopy (Editorial)**—Wenzel RP, Edmond MB. JAMA 1997;278(13):1111.

Global Theme Issue on Aging. Chest 1997;112(4), October. A series of articles devoted to cardiopulmonary and critical care issues of the aging, published in conjunction with 90 other journals from around the world.

Recent Transmission of Tuberculosis in a Cohort of HIV-1-Infected Female Sex Workers in Nairobi, Kenya—Gilks CF, Godfrey-Faussett P, Batchelor BI, Ojoo JC, Ojoo SJ, Brindle RJ, et al. AIDS 1997;11(7):911.

OBJECTIVES: To describe the epidemiological and clinical characteristics of HIV-related tuberculosis in a female cohort and to investigate the relative importance of recently transmitted infection and reactivation in the pathogenesis of adult HIV-related tuberculosis. DESIGN: Members of an established cohort of female sex workers in Nairobi were enrolled in a prospective study. Women were followed up regularly and seen on demand when sick. METHODS: Between October 1989 and September 1992 we followed 587 HIV-infected and 132 HIV-seronegative women. Standard protocols were used to investigate common presentations. Cases of tuberculosis were identified clinically or by culture. All available *Mycobacterium tuberculosis* strains underwent DNA fingerprint analysis. RESULTS: Forty-nine incident and 4 recurrent episodes of tuberculosis were seen in HIV-infected women; no disease was seen in seronegative sex workers ($p = 0.0003$). The overall incidence rate of tuberculosis was 34.5/1000 person-years amongst HIV-infected participants. In purified protein derivative (PPD) skin test-positive women the rate was 66.7/1000 person-years versus 18.1/1000 person-years in PPD-negative women. Twenty incident cases (41%) were clinically compatible with primary disease. DNA fingerprint analysis of strains from 32 incident cases identified 2 clusters comprising 2 and 9 patients; allowing for index cases, 10 patients (28%) may have had recently transmitted disease. Three out of 10 (30%) patients who were initially PPD skin test-negative became PPD-positive. Taken together, 26 incident cases (53%) may have been recently infected. DNA fingerprint analysis also identified 2 (50%) of the 4 recurrent tuberculosis episodes as reinfection. CONCLUSIONS: Substantial recent transmission of tuberculosis appears to be occurring in Nairobi amongst HIV-infected sex workers. It may be incorrect to assume in other regions of high tuberculosis transmission that active HIV-related tuberculosis usually represents reactivation of latent infection.

Dust-Mite-Allergen Concentrations in Asthmatics' Bedrooms in the Quad Cities (Illinois, USA) after the Mississippi River Floods of 1993—Curtis L, Ross M, Scheff P, Persky V, Wadden R, Ramakrishnan V, Hryhorczuk D. Allergy 1997;52(6):642.

This study aimed to measure allergens from *Dermatophagoides farinae* and *D. pteronyssinus* and to examine possible relationships of these mite allergens with flooding and other housing factors. A total of 313 dust samples were collected from the bedrooms of 57 asthmatics in 45 homes in the Quad Cities (Illinois, USA) and analyzed by ELISA for the presence of the *D. farinae* and *D. pteronyssinus* allergens. Twenty of these homes had some flooding in the last 12 months due to the Mississippi River floods of 1993 and/or other factors. The log-transformed least-squares means of allergens collected were 28 ng/m^2 for the *D. farinae* allergen and 26 ng/m^2 for the *D. pteronyssinus* allergen. *D. farinae* allergen levels were significantly higher in homes located in the valley, in homes during the summer months, in homes with furred or feathered pets, in homes which had not been flooded in the last year, and in homes where rugs had been steam-cleaned in the last 12 months. *D. pteronyssinus* allergen levels were significantly higher in homes located in the valley, in homes during April and July-September, in homes with furred or feathered pets, and in homes with no dehumidifier.

Bronchiolitis Obliterans and No Radiographic Abnormalities in a Patient with Rheumatoid Arthritis—Kagamu H, Hasegawa T, Kurashige K, Saitoh Y, Terada M, Satoh M, et al. Nippon Kyobu Shikkan Gakkai Zasshi 1997;35(5):524.

A 53-year-old woman was given a diagnosis of rheumatoid arthritis in 1988, and began treatment with D-penicillamine in September 1992. She noticed dry coughing and exertional dyspnea that began in April 1993. Chest x-ray and CT films revealed no abnormal opacities. However, bronchiolitis obliterans was suspected because of a low FEV_1% (23%). Examination of specimens obtained by thoracoscopic lung biopsy revealed constrictive obliteration by granulation tissue in

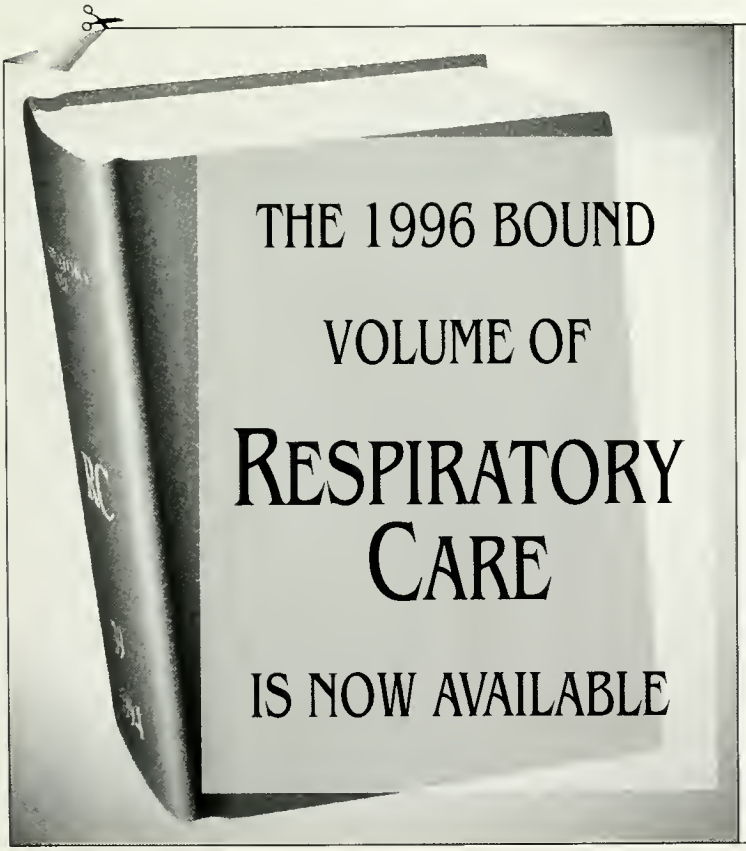
proximal bronchioles and follicular bronchiolitis. Alveoli and respiratory bronchioles were intact. After corticosteroid and cyclophosphamide pulse therapy, FEV_1% increased to 35%. At the time of this writing she was alive 2.5 years after hospitalization.

Measuring the Quality of Health Care: State of the Art—Donaldson MS, Nolan K. Jt Comm J Qual Improv 1997;23(5):283.

BACKGROUND: September 12-13, 1996, in Washington, DC, the Institute of Medicine, as part of its Special Initiative on Health Care Quality, held an invitational conference to illustrate exemplary quality measurement and to discuss the results. Patient reports, innovative uses of outcome measures for quality improvement, risk adjustment, assessment in integrated health plans and health care settings, clinical guidelines, and projects on disseminating information on quality measurement techniques and tools were among the topics represented. IMPROVING MEDICATION USE: Brent James described studies undertaken to reduce adverse drug events (ADEs). When implementing any measurement system where error is a possible factor, it is important to emphasize identifying problems for the purpose of learning, not judgment. Lucian Leape agreed that staff involved must feel that the purpose of the study is to examine system problems, not individuals' mistakes. David Classen described a nonproprietary computerized disease-management program designed to reduce ADEs in infectious diseases. A QUALITY VISION: Robert Brook said that the relationship between cost or resources devoted to care and quality is not well understood and is certainly not simple. He also said that although investments in measurement strategies are needed to make them better, that does not mean we should not attempt to use the measurements we have now. CLOSING ADDRESS: Mark Chassin said that the presentations at the conference provided evidence that should allow us to conclude beyond a reasonable doubt that quality can be measured—with a degree of scientific precision equal to that of most of the measures used to take care of patients every day.

Status of Lung Transplant Recipients Surviving Beyond Five Years—Chaparro C, Scavuzzo M, Winton T, Keshavjee S, Kesten S. J Heart Lung Transplant 1997;16(5):511.

BACKGROUND: Prolonged survival after lung transplantation is now commonplace as a result of advances in surgical techniques and postoperative management protocols. Although 1- and 5-year functional and survival data after lung transplantation are well known, sparse information is available regarding functional status of recipients surviving beyond 5 years. METHODS: The medical records and pulmonary function study results of lung transplant recipients who had survived at least 5 years as of September 1995 were retrospectively reviewed. RESULTS: Of the 76 transplantations performed between November 1983 and September 1990, 30 (39.5%) were double lung transplantations, and 46 (60.5%) were single lung transplantations. Thirty-one recipients were alive 5 years after transplantation (12 double lung transplantations, 19 single lung transplantations). The 5-, 6-, and 7-year survival rates were 44%, 34%, and 29%, respectively. There was no association or difference in cytomegalovirus status, sex, and blood group between those who died within 5 years and those who survived beyond 5 years. The median percent predicted FEVs for single and double lung transplant recipients were as follows: 5 years—75%, 75%; 6 years—73%, 75%; 7 years—68%, 73%. The proportion of recipients with bronchiolitis obliterans syndrome according to published criteria was as follows: Stage 0, 32%; Stage I, 19%; Stage II, 16%; and Stage III, 19%. The functional status (ie, active, working, disabled) 5 years after transplantation was as follows: active/working, 74%; active but not working, 13%; some limitation/independent, 10%; and disabled, 3%. CONCLUSION: Bronchiolitis obliterans syndrome is a frequent occurrence in long-term sur-



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vivors. Nevertheless, in spite of this condition, most recipients have acceptable lung function, are active, and are generally working.

Lung Transplantation: The Experience of the Thoracic Organ Transplantation Centre of Pavia—Fracchia C, Callegari G, Volpato G, Martinelli L, Rinaldi M, Rampulla C, Ambrosino N, Monaldi Arch Chest Dis 1997;52(2):126.

Between January 1991 and September 1995 at the Thoracic Organ Transplantation Centre of Pavia, 193 patients entered the waiting list for heart-lung or lung transplantation. Indications for heart-lung transplantation (HLT) were mainly primary or secondary pulmonary vascular diseases. Parenchymal lung diseases were the most frequent reasons for single- (SLT) or double-lung (DLT) transplantation. During the same period, 21 patients underwent HLT, 16 SLT, and 14 DLT. Early deaths (within 30 days of surgery) occurred in 2 (10%) HLT, in 3 (19%) SLT, and in 3 (21%) DLT. Nineteen (90%) patients with HLT, 11 (69%) with SLT, and 10 (71%) with DLT survived up to 3 months; and 11 (52%) patients with HLT, 8 (50%) with SLT, and 5 (36%) with DLT survived up to 12 months. At the time of writing, the following patients are still alive: 10 (48%) with HLT, after a mean ± SEM follow-up of 37.2 ± 6 (range 28-46) months, 12 (75%) with SLT, after a mean follow-up of 16 ± 11 (range 1-35) months, and finally 7 (50%) with DLT, after mean follow-up of 14 ± 9 (range 1-23) months.

Understanding the Quality Challenge for Health Consumers: The Kaiser/AHCPR Survey—Robinson S, Brodie M. Jt Comm J Qual Improv 1997;23(5):239.

BACKGROUND & PURPOSE: "Americans as Health Care Consumers: The Role of Quality Information," a nationally representative telephone survey of 2,006 adults, designed by the Henry J Kaiser Family Foundation (Kaiser) and the Agency for Health Care Policy and Research (AHCPR) of the U.S. Department of Health and Human Services, was presented at the Kaiser/AHCPR conference "Value and Choice: Providing Consumers with Information on the Quality of Care" in Arlington, Virginia, October 29-30, 1996. The survey was conducted by Princeton Survey Research Associates between July 16 and September 5, 1996 and has a margin of error of ±3 percentage points. RESULTS: The survey found that Americans value quality but that the use of quality indicators is likely to be only 1 factor in their decision making, given their reliance on and preference for quality information from their physicians, friends, and family. A plurality (42%) chose having high quality of care as their most important concern, but only 39% reported seeing information comparing health plans, and only about one third of that group said they used this information themselves to make decisions. The following are possible explanations for why consumers don't use information on quality in making health care decisions: They trust friends, families, and physicians about all other sources for advice; They do not (at the time of the survey) have many health plan choices to make; or they lack familiarity with comparative health care quality information.

One Hundred Video-Assisted Thoracic Surgical Simultaneously Stapled Lobectomies without Rib Spreading—Lewis RJ, Caccavale RJ, Sisler GE, Bocage JP, Mackenzie JW. Ann Thorac Surg 1997;63(5):1415.

BACKGROUND: This study was performed to evaluate and determine the validity and benefits of video-assisted thoracic surgical simultaneously stapled pulmonary lobectomy without rib spreading. METHODS: Between September 1992 and August 1995, 100 consecutive video-assisted thoracic surgical simultaneously stapled lobectomies without rib spreading were performed. RESULTS: Forty-five male and 55 female patients had 24 right upper, 8 right middle, 29 right lower, 24 left upper, 15 left lower lobectomies for 66 adenocarcinomas, 20 squamous cell carcinomas, 4 large cell carcinomas, 8 benign lesions, and 2 metastatic lesions. Seventy-six patients had negative nodes. Nine patients had positive nodes. Every bronchoscopy was visually and cytologically negative. Forty-nine cervical mediastinoscopies were negative. Operating time for the series averaged 90.3 minutes. Hospitalization averaged 3.5 days for the entire group, but was 2.6 days for the last 20 patients. Lesions ranged from 1.5 to 8 cm, averaging 3.4 cm. There was no surgical mortality, no hemorrhage, no transfusion, and no urgent conversion to an open procedure. No bronchial fistula, vascular fistula, or bronchovascular fistula has occurred. Complications included 6 air leaks, 2 cerebrovascular accidents, 1 infected chest tube site, 2 cases of pneumonitis, and 1 subcutaneous emphysema. CONCLUSIONS: Video-assisted thoracic surgical simultaneously stapled lobectomy without rib spreading is a safe operation that can be combined with lymph node sampling. At this early stage, therapeutic outcomes (survival) for resected neoplasms appear similar to results obtained from traditional open techniques.

Effects of Ambient Ozone Exposures during the Spring and Summer of 1994 on Pulmonary Function of Schoolchildren—Ulmer C, Kopp M, Ihorst G, Frischer T, Forster J, Kuehr J. Pediatr Pulmonol 1997;23(5):344.

To investigate the effect of natural exposure to ambient ozone over time, a follow-up study of school-aged children was performed in 2 small towns in southwestern Germany (Freudenstadt and Villingen) between March and October of 1994. Ozone half-hour mean concentrations were measured continuously and pulmonary function was tested in each child on 4 occasions (April, June, August, and September). To obtain an average short-term ozone effect, we first analyzed the data from the 4 time points separately and then constructed a model that included all information. During the study period the median (5th to 95th percentile) of all half-hour values of the ozone concentration was 101 $\mu g/m^3$ or 50.6 ppb (45-179 $\mu g/m^3$ or 22.5-89.8 ppb) in Freudenstadt and 64 $\mu g/m^3$ or 32.1 ppb (1 to 140 $\mu g/m^3$ or 0.5-70.1 ppb) in Villingen. To assess the effects of an individual ozone exposure we related the highest ozone concentration in the respective 24 hours before lung function testing to the results of the subconcentration in the respective 24 hours before lung function testing to the results of the subsequent pulmonary function tests. In the lung function test following the highest ozone exposure, the results of our cross-sectional linear regression analysis showed a significant negative correlation ($p = 0.0181$) between ozone exposure and forced vital capacity (FVC). In the longitudinal linear regression model we observed a negative statistical correlation between ozone exposure and lung function for the subpopulation living in the town with the high ozone levels (Freudenstadt). The association was more pronounced in boys than girls. For the children in Freudenstadt the decrement of FVC was −12.31 mL/10 $\mu g/m^3$ ozone and the decrease in the forced expiratory volume in 1 second (FEV_1) was −11.29 mL/10 $\mu g/m^3$ ozone.

Indications for Flexible versus Rigid Bronchoscopy in Children with Suspected Foreign-Body Aspiration—Martinot A, Closset M, Marquette CH, Hue V, Deschildre A, Ramon P, et al. Am J Respir Crit Care Med 1997;155(5):1676.

Diagnostic indications for flexible bronchoscopy in the initial investigation of children with suspected foreign-body (FB) aspiration have not been evaluated prospectively. We prospectively collected history, clinical, and radiologic findings at prebronchoscopic examination of all children referred for suspected FB aspiration between February 1993 and September 1995. Children with asphyxiating FB aspiration, requiring immediate rigid bronchoscopy, were excluded. If there was clear evidence of FB aspiration from the physical and radiographic findings, rigid bronchoscopy was directly performed. If the evidence was not convincing, children underwent diagnostic flexible bronchoscopy under local anesthesia. If an FB was found, rigid bronchoscopy was always performed for extraction. Eighty-three consecutive children (median age: 24 mo) were included. Among 28 who underwent rigid bronchoscopy first, 23 had an FB. Among the 55 children who underwent flexible bronchoscopy first, 17 had an FB. Predictive signs of a bronchial FB were a radiopaque FB, and associated unilaterally decreased breath sounds and obstructive emphysema (positive predictive value = 0.94). We propose the following management algorithm: Rigid bronchoscopy is performed first in case of as-

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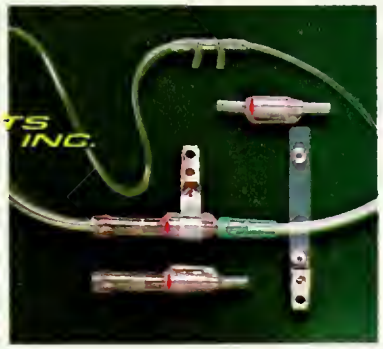

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phyxia, a radiopaque FB, or association of unilaterally decreased breath sounds and obstructive emphysema. In any other case, flexible bronchoscopy is performed first for diagnostic purposes. If applied retrospectively to the 83 children in our study, this algorithm would have decreased the negative first rigid bronchoscopy rate to 4%. Flexible bronchoscopy is a safe and cost-saving diagnostic procedure in children with suspected FB aspiration.

Association of *Sauropus Androgynus* and Bronchiolitis Obliterans Syndrome: A Hospital-Based Case-Control Study—Ger LP, Chiang AA, Lai RS, Chen SM, Tseng CJ. Am J Epidemiol 1997 May 1;145(9):842.

In late April 1995, an outbreak of a poorly defined respiratory illness related to the ingestion of leaves of *Sauropus androgynus* was observed in southern Taiwan. To further evaluate the association between *S. androgynus* and bronchiolitis obliterans syndrome, a hospital-based case-control study was conducted with 1 case group and 3 different control groups at Veterans General Hospital-Kaohsiung between April and September 1995. A total of 54 cases (50 females, 4 males), 54 age- and sex-matched neighborhood controls, 54 matched routine physical check-up controls, and 54 matched self-referred patron controls (who had ingested *S. androgynus* yet without obstructive physiology) were interviewed for clinical symptoms, history of *S. androgynus* consumption, and potential confounding factors. All 54 cases (100%) ingested *S. androgynus* compared with only 5 (9%) neighborhood controls (matched odds ratio (OR) incalculable, $p < 0.001$) and 2 (4%) physical check-up controls (matched OR incalculable, $p < 0.001$). In the univariate analysis of 54 cases and 54 self-referred patron controls, factors associated with an increased risk of bronchiolitis obliterans syndrome were methods of food preparation (uncooked juice vs stir fried or boiled dishes, matched OR 10.3 (95% confidence interval [CI] 1.3-84.4); preparer of the *S. androgynus*-containing food (vendor only vs patient only or patient plus vendor, matched OR 2.8 (95% CI 1.1-7.1); total *S. androgynus* consumption quantity (> 4,500 vs 413-2,063 g, matched OR 10.0 [95% CI 1.9-53.0]); duration of consumption (> 45 vs 6-24 days, matched OR 2.1 [95% CI 1.2-3.8]); and midterm interruption (< 2 vs 2-5 days/week, matched OR 2.6 [95% CI 1.1-6.1]). Additionally, multiple conditional logistic regression analysis of cases and self-referred patron controls revealed that a larger total amount of *S. androgynus* consumption, preparation of *S. androgynus* food without cooking, and ingesting *S. androgynus* food prepared by a vendor were the significant risk factors associated with bronchiolitis obliterans syndrome.

Physician Based Surveillance System for Occupational Respiratory Diseases: The Experience of PROPULSE, Quebec, Canada—Provencher S, Labreche FP, De Guire L. Occup Environ Med 1997;54(4):272.

OBJECTIVE: To evaluate the feasibility of implementing a physician-based surveillance system of occupational respiratory diseases (PROPULSE) in Quebec with regard to physician participation rate, characteristics of reported cases, and comparison with official statistics from the Workers' Compensation Board (WCB). METHODS: All chest physicians and allergists in Quebec were asked to report suspected new cases of occupational respiratory diseases, on a monthly basis, between October 1992 and September 1993. For each case, personal information was collected and the physician's opinion on whether the condition was related to work was categorised as highly likely, likely, and unlikely. RESULTS: Of the 161 physicians initially approached, 68% participated. Physicians rated 48% of suspected cases as highly likely, 29% as likely, and 20% as unlikely. The most often reported diagnosis was asthma (63%), followed by diseases related to asbestos (16%). Silicosis was less frequent (5%) but it was reported for 6 workers under 40 of whom 5 were involved in sandblasting activities. The high proportion of cases of asthma probably reflects the increasing importance of this disease but may also reflect the different patterns of reporting among physicians with different expertise. The distribution of cases by diagnostic category is quite different between the PROPULSE system and that of the WCB (annual mean number of compensated cases during a 4-year period). Asthma and allergic alveolitis are more frequent in PROPULSE, reactive airways dysfunction syndrome are about the same in both systems, and other diseases are more frequent among compensated cases. The most frequent sensitising agents reported for asthma were the same in both systems (isocyanates, flour, and wood dust). 15% of the PROPULSE cases were not covered by the WCB, and therefore would not be found in the board's official statistics. CONCLUSIONS: A physician based reporting procedure can be implemented as part of a surveillance system to supplement data from other sources and thus provide a better understanding of the occurrence of occupational respiratory diseases.

Chlamydia Pneumoniae as a New Source of Infectious Outbreaks in Nursing Homes—Troy CJ, Peeling RW, Ellis AG, Hockin JC, Bennett DA, Murphy MR, Spika JS. JAMA 1997;277(15): 1214. [Published erratum appears in JAMA 1997;278(2):118].

OBJECTIVE: To determine the extent and severity of illness and mode of transmission of *Chlamydia pneumoniae* infection in 3 nursing home outbreaks. DESIGN & SETTING: Retrospective cohort study in 3 nursing homes in Ontario from September to November 1994. SUBJECTS: A total of 549 residents and 65 staff members. MAIN OUTCOME MEASURES: Morbidity and mortality were determined by a review of disease surveillance forms, residents' charts, and a self-administered questionnaire to staff. Single and paired serum samples for *C. pneumoniae* serological testing and nasopharyngeal swabs for *C. pneumoniae* culture were collected, and direct fluorescent antibody assays were performed to confirm *C. pneumoniae* infection. RESULTS: The attack rates for confirmed and suspected cases combined were 68%, 46%, and 44% among residents in nursing homes A, B, and C, respectively, and 34% among nursing home C staff. A total of 16 cases of pneumonia confirmed by chest x-ray and 6 deaths were identified. The spectrum of illness among nursing home C residents included a new cough in 58 (100%), fever in 37 (64%), sore throat in 14 (24%), and hoarseness in 8 (14%). Staff members at nursing home C were more likely to report hoarseness ($p < 0.001$) and sore throat ($p < 0.001$). Residents who smoked had onset of illness earlier than nonsmokers ($p = 0.007$), which perhaps is related to airborne transmission in a designated smoking room. CONCLUSIONS: *Chlamydia pneumoniae* caused serious morbidity and mortality among residents and morbidity among staff; *C. pneumoniae* is an important cause of respiratory disease outbreaks in nursing homes, and diagnostic tests must be readily available for early recognition of *C. pneumoniae* infections.

Cost Analysis and Clinical Impact of Weekly Ventilator Circuit Changes in Patients in Intensive Care Unit—Kotilainen HR, Keroack MA. Am J Infect Control 1997;25(2):117.

BACKGROUND: The introduction of heated circuits and sealed, single-use humidifiers has prompted some investigators to question the traditional recommendations for changing ventilator circuits. We studied the clinical and cost impact of extending the circuit change interval from 72 hours to 7 days in our 2 intensive care units with 17 beds. METHODS: With standard surveillance definitions from the Centers for Disease Control and Prevention and the National Nosocomial Infections Surveillance System, baseline pneumonia rates were established for a 3-month period. After the institution of weekly circuit changes, daily surveillance of intubated patients was performed during 18 of 22 weeks from May through September 1993. Standard microbiologic methods were used for the identification of patient and environmental isolates. RESULTS: Ventilator-associated pneumonia for the 72-hour circuit change group was 9.1% or 1.29/100 ventilator days. After the institution of weekly changes, pneumonia occurred in 9 of 146 patients (6.2% or 0.74/100 ventilator days χ^2 = 0.33, p = 0.44). No common bacterial isolates were recovered as judged by phenotype, biochemical, or antimicrobial susceptibility patterns. Weekly changes reduced the number of circuits used from a predicted 469 to 214. Estimating $26.46/circuit change, annualized cost savings were $20,246.90. CONCLUSIONS: Weekly circuited changes in patients undergoing ventilation therapy in the intensive care unit are cost-effective and do not contribute to increased rates of nosocomial pneumonia.

Socioeconomic Status and Lung Cancer Incidence in Men in the Netherlands: Is There a Role for Occupational Exposure?—van Loon AJ, Goldbohm RA, Kant IJ, Swaen GM, Kremer AM, van den Brandt PA. J Epidemiol Community Health 1997;51(1):24.

OBJECTIVE: To evaluate the influence of occupational exposure to carcinogens in explaining the association between socioeconomic status and lung cancer. DESIGN: A prospective cohort study. Data on diet, other lifestyle factors, sociodemographic characteristics, and job history were collected by means of a self-administered questionnaire. Follow-up for incident cancer was established by record linkage with a national pathology register and with regional cancer registries. SETTING: Population originating from 204 municipalities in The Netherlands. PARTICIPANTS: These comprised 58,279 men aged 55-69 years in September 1986. After 4.3 years of follow-up there were 470 microscopically confirmed incident lung cancer cases with complete data on dietary habits and job history. MEASUREMENTS & MAIN RESULTS: Estimation of occupational exposure to asbestos, paint dust, polycyclic aromatic hydrocarbons, and welding fumes was carried out by 2 experts, using information on job history from the baseline questionnaire. Socioeconomic status was measured by means of highest attained level of education and 2 indicators based on occupation. In the initial multivariate analyses of socioeconomic status and lung cancer, adjustment was made for age, smoking habits, intake of vitamin C, β-carotene, and retinol, and history of chronic obstructive pulmonary disease or asthma. Additional adjustment for occupational exposure to the 4 carcinogens mentioned above did not change the inverse association between the level of education and lung cancer risk (initial model: RR highest/lowest level of education = 0.53; 95% CI 0.34, 0.82; additional model: RR highest/lowest level of education = 0.53; 95% CI 0.34, 0.84). Nor was the association between the 2 occupation based indicators of socioeconomic status and lung cancer risk influenced by occupational exposure to carcinogens. The effect of occupational exposure on the association between the level of education and lung cancer risk did not differ between ex-smokers and current smokers. CONCLUSIONS: Occupational exposure to asbestos, paint dust, polycyclic aromatic hydrocarbons, and welding fumes could not explain the inverse association between socioeconomic status and lung cancer risk. More research which explicitly addresses possible explanations for the association between socioeconomic status and lung cancer risk is needed.

Under the Volcano: Fire, Ash and Asthma?—Bradshaw L, Fishwick D, Kemp T, Lewis S, Rains N, Slater T, et al. N Z Med J 1997;110(1040):90.

AIMS: To investigate the prevalence of respiratory symptoms in known asthmatics, following exposure to airborne volcanic ash particles caused by the eruptions of Mount Ruapehu, New Zealand, commencing September 1995. METHOD: A 1-page postal questionnaire was sent to 1392 previously identified asthmatics 2 months after the first major eruption. RESULTS: Two hundred and thirty-seven subjects had moved from the area, died, or gone overseas since the original contact 4 years previously; therefore the target population was 1155 subjects of whom 361 lived in the exposed area and 794 in the nonexposed areas. The response rates were 246 (68.1%) in the exposed group and 477 (60.1%) in the nonexposed group making a total of 723 individuals. The prevalence of nocturnal shortness of breath in the last 2 months was 29.3% in the exposed group and 24.7% in the nonexposed (OR = 1.26, 95% CI; 0.83-1.78). Similarly 30.9% of the exposed group had an asthma attack in the last 2 months compared to 31.9% of the nonexposed group (OR = 0.96, 95% CI; 0.69-1.33). Finally, 48.4% of the exposed group used asthma medication in the 2 months following the eruption in comparison to 53% of the nonexposed group (OR = 0.83, 95%, CI; 0.61-1.12). CONCLUSIONS: The study showed no association between living in an area exposed to volcanic ash particles and either asthma symptoms or the use of asthma medication. There was a small but nonsignificant increase in nocturnal shortness of breath in the exposed group.

Inhaled Steroids and the Risk of Hospitalization for Asthma—Donahue JG, Weiss ST, Livingston JM, Goetsch MA, Greineder DK, Platt R. JAMA 1997;277(11):887.

OBJECTIVE: To determine if anti-inflammatory treatment for asthma reduces the risk of asthma hospitalization. DESIGN: Retrospective cohort study. SETTING: A health maintenance organization (HMO) in eastern Massachusetts. PARTICIPANTS: Members of the HMO who were identified during the period October 1991 through September 1994 as having a diagnosis of asthma using a computerized medical record system. MAIN OUTCOME: Hospitalization for asthma. RESULTS: Of the 16,941 eligible persons, 742 (4.4%) were hospitalized for asthma. The overall relative risk (RR) of hospitalization among those who received inhaled steroids was 0.5 (95% confidence interval [CI], 0.4-0.6) after adjustment for β-agonist dispensing. Additional adjustment for age, race, other asthma medications, and amount and type of ambulatory care for asthma did not substantially affect the inverse relationship between use of inhaled steroids and hospitalization. Cromolyn was similarly associated with reduced risk, especially among children (RR, 0.8; 95% CI, 0.7-0.9). In contrast, increasing β-agonist use was associated with increasing hospitalization risk even after adjustment for other factors and medications. The steroid-associated protection was most marked among individuals who received the largest amount of β-agonist. CONCLUSIONS: Inhaled steroids and, to a lesser extent, cromolyn confer significant protection against exacerbations of asthma leading to hospitalization. These results support the use of inhaled steroids by individuals who require more than occasional β-agonist use to control asthma symptoms.

Relapse of Tuberculosis after Treatment in Human Immunodeficiency Virus-Infected Patients—Pulido F, Pena JM, Rubio R, Moreno S, Gonzalez J, Guijarro C, et al. Arch Intern Med 1997;157(2):227.

OBJECTIVES: To evaluate the relapse rate of tuberculosis after a complete course of antituberculous therapy in human immunodeficiency virus-infected patients and to identify the risk factors for relapse. PATIENTS & METHODS: Historic cohort study of all adult patients who were diagnosed as having human immunodeficiency virus infection and a first episode of culture-proved tuberculosis at 2 university hospitals in Madrid,

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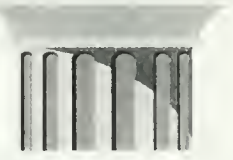

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Spain, between 1986 and 1992, and who completed at least 6 months of treatment were included and followed up until September 1994. RESULTS: Of 276 patients with human immunodeficiency virus infection and tuberculosis, 87 could not be evaluated (6 died before treatment, 39 died during treatment, 36 did not complete the planned therapy, and 6 were unavailable during treatment). The remaining 189 received a standard regimen (ie, 3 or 4 drugs, always including rifampin and isoniazid, for ≥ 6 months). The median duration of follow-up for these 189 patients was 31.5 months, with a total of 4668 patient-months of follow-up after treatment; 105 patients (56%) were followed up until death. The relapse rate was 7.9% (2.7/100 patient-years). With multivariate analysis, a shorter duration of treatment and a low CD4+ cell count were associated with a greater probability of relapse. Relapses occurred in 5 (3.4%) of 148 patients who were treated for 9 or more months (1.7/100 patient-years) and in 10 (24%) of 41 patients who were treated for less than 9 months (10.9/100 patient-years) ($p < 0.001$; relative hazard, 9.2; 95% confidence interval, 3.1-26.9). CONCLUSIONS: As standard antituberculous therapy for 9 months is associated with a low rate of relapse, maintenance therapy is not required. Duration of treatment for <9 months is associated with a high rate of relapse.

Detection of Methicillin-Resistant *Staphylococcus Aureus* (MRSA)—Relation between Respiratory Tract and Gastro-Intestinal Tract—Ito Y, Tanaka M, Shimazaki M, Nakamura T, Kimura Y, Shima H, et al. Kansenshogaku Zasshi 1997;71(3):207.

The study was conducted to elucidate the possibility of hospital infection of methicillin-resistant *Staphylococcus aureaus* (MRSA) through feces. Fecal cultures of 12 inpatients positive for MRSA in the respiratory tract and 11 inpatients negative for MRSA in the respiratory tract were performed from April to October in 1993. Fecal cultures of inpatients positive for MRSA in the respiratory tract were continued until September in 1996, finally a total of 50 cases were enrolled. MRSA was isolated from feces of 7 patients (58.3%) of the 12 patients positive for MRSA in the respiratory tract, while no MRSA was isolated from feces of 11 patients negative for MRSA in the respiratory tract. Twenty-seven patients (54.0%) of 50 patients positive for MRSA in the respiratory tract yielded MRSA in the feces. Twenty-three patients (85.2%) of the 27 patients positive for MRSA in the fecal flora had received H2-blocker, while 11 patients (47.8%) of the 23 patients negative for MRSA in the fecal flora received H2-blocker; these differences were statistically significantly ($p < 0.01$). These findings suggest the possible role of feces in hospital infection with MRSA and the necessity for careful administration of H2-blocker to patients positive for MRSA in the respiratory tract.

Efficacy of Atropine Sulfate in Combination with Albuterol in the Treatment for Acute Asthma—Diaz JE, Dubin R, Gaeta TJ, Pelczar P, Bradley K. Acad Emerg Med 1997;4(2):107.

OBJECTIVE: To determine the efficacy of combination therapy using atropine sulfate and albuterol in the treatment for an acute exacerbation of asthma. METHODS: A prospective, randomized double-blind, placebo-controlled study was performed in the ED of a large, inner-city, university-affiliated teaching hospital. Participants were a convenience sample of patients presenting to the ED between September 1993 and March 1994 with acute exacerbations of their asthma. Patients judged to be in extremis were excluded. All patients received 3 nebulized treatments with 2.5 mg of albuterol at 0, 30, and 60 minutes. Patients were randomized into 1 of 3 groups with the following added to their nebulizer solutions: (1) saline placebo during all 3 treatments; (2) 2.0 mg atropine sulfate added to the first nebulizer and saline

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in the second and third; or (3) 2.0 mg atropine to the first and third treatments (with saline in the second). No other medication was administered during the study period. At 90 minutes, the patients were evaluated for admission or release from the ED according to predetermined criteria, and additional medications were given as necessary. Vital signs, peak expiratory flow (PEFR), degree of wheezing, level of distress, and side effects were measured before and after each nebulizer treatment. RESULTS: Of the 153 patients eligible for the study, 126 completed the entire study protocol. There was no significant difference between the 3 groups on any parameter studied, including improvement of PEFR, vital signs, or level of distress. There was no difference in the admission rate between the 3 groups, nor was there a difference in the incidence of side effects among the groups. CONCLUSION: In this study population, combination therapy with atropine sulfate and albuterol offered no significant benefit over the use of albuterol alone in the treatment for acute exacerbation of asthma.

Effects of Air Pollution on Emergency Room Visits for Respiratory Illnesses in Montreal, Quebec—Delfino RJ, Murphy-Moulton AM, Burnett RT, Brook JR, Becklake MR. Am J Respir Crit Care Med 1997;155(2):568.

As an approach to evaluating the public health burden from current air pollution levels, we examined the relationship of daily emergency room (ER) visits for respiratory illnesses (25 hospitals, average 98 visits/d) to air pollution in Montreal, Canada, from June through September 1992 and 1993. Air pollutants measured included ozone (O_3), particulate matter diameter < 10 μm (PM10) and < 2.5 μm (PM2.5), the sulfate fraction of PM2.5 (SO_4), and aerosol strong acidity (H^+). Temporal trends, autocorrelation, and weather were controlled for in time-series regressions. For 1992, no significant associations with ER visits were found. However, 33% of the particulate data were missing. For 1993, 1-hour maximum O_3, PM10, PM2.5, and SO_4 were all positively associated with respiratory visits for patients over 64 years of age ($p < 0.02$). An increase to the mean level of 1-hour maximum O_3 (36 ppb) was associated with a 21% increase over the mean number of daily ER visits (95% confidence interval [CI]: 8 to 34%). Effects of particulates were smaller, with mean increases of 16% (4 to 28%), 12% (2 to 21%) and 6% (1 to 12%) for PM10, PM2.5, and SO_4, respectively. Relative mass effects were PM2.5 > PM10 > SO_4. Ozone and PM10 levels never exceeded 67 ppb and 51 $\mu g/m^3$, respectively (well below the U.S. National Ambient Air Quality Standards of 120 ppb and 150 $\mu g/m^3$, respectively). The present findings have public health implications with regard to the adverse health effects of urban photochemical air pollution on older individuals.

Dose-Intensive Chemotherapy in Extensive-Stage Small-Cell Lung Cancer—Negoro S, Masuda N, Furuse K, Saijo N, Fukuoka M. Cancer Chemother Pharmacol 1997;40 Suppl:S70.

The importance of the dose intensity of chemotherapy in achieving maximal therapeutic effect has recently been reported for several chemosensitive malignant diseases, with Murray et al reporting that intensive weekly chemotherapy using the cisplatin, vincristine, doxorubicin, and etoposide (CODE) regimen in small-cell lung cancer (SCLC) is very effective. However, leukopenia is the major obstacle to delivering the planned dose in intensive regimens. Therefore, we investigated whether recombinant human granulocyte colony-stimulating factor (rhG-CSF) could allow full drug doses to be given as scheduled, thereby improving the final outcome. Extensive-stage (ES) SCLC patients were randomized to receive either CODE alone or CODE with rhG-CSF, with the CODE regimen consisting of cisplatin given I.V. at 25 mg/m^2 weekly for 9 weeks; vincristine given I.V. at 1 mg/m^2 during Weeks 1, 2, 4, 6, and 8; and doxorubicin given I.V. at 40 mg/m^2 and etoposide given I.V. at 80 mg/m^2 for 3 days during Weeks 1, 3, 5, 7, and 9, rhG-CSF at 50 μg/m2 was given s.c. on the days on which cytotoxic drugs were not given. From May 1989 to September 1991; 64 patients were enrolled in the study, of whom 63 were analyzable (31 for CODE alone and 32 for CODE with rhG-CSF). No difference in any of the patients' characteristics except gender was found between the 2 groups. The complete response (CR) rate was 34% in the CODE with rhG-CSF group and 23% in the CODE alone group; the median survival was 59 and 32 weeks, respectively, in these groups ($p = 0.004$). Therefore CODE with rhG-CSF improved the survival of ES SCLC patients. On the basis of these results a Phase-III study to determine whether CODE with rhG-CSF would increase survival as compared to the standard regimen in ES SCLC was designed by the Japan Clinical Oncology Group.

Combined Heart-Lung-Liver, Double Lung-Liver, and Isolated Liver Transplantation for Cystic Fibrosis in Children—Couetil JP, Soubrane O, Houssin DP, Dousset BE, Chevalier PG, Guinvarch A, et al. Transpl Int 1997;10(1):33.

Between June 1990 and September 1995, 8 of 24 children with cystic fibrosis (CF) who were accepted either for combined transplantation or isolated liver transplantation died while waiting for a graft; 11 underwent transplantation, and 5 are currently on the waiting list. Of the 11 children who had surgery, 7 (Group 1) underwent one of the following procedures: heart-lung-liver (n = 4), sequential double lung-liver (n = 2), or bilateral lobar lung from a split left lung and reduced liver (n = 1). During the same period, the 4 other children (Group 2) underwent isolated liver transplantation (3 full-size livers, 1 partial liver). There was 1 perioperative death in each group. Pulmonary infection was the most common cause of morbidity in Group 1. Other complications in Group 1 included tracheobronchial stenosis (n = 2), biliary stricture (n = 2), and severe ascites (n = 2). All were successfully treated. Obliterative bronchiolitis developed in 3 patients. This was treated with FK 506. In Group 2, pulmonary function tests improved or remained stable after liver transplantation. Surgical complications in Group 2 included severe ascites (n = 1), biliary stricture (n = 1), and abscess of the liver (n = 1). Actuarial survival was 85.7% ± 2% in Group 1 at 1 year; it remained unchanged at 3 years and was 64.2% at 5 years.

A Nosocomial Outbreak of Multidrug-Resistant Tuberculosis—Kenyon TA, Ridzon R, Luskin-Hawk R, Schultz C, Paul WS, Valway SE, et al. Ann Intern Med 1997;127(1):32.

BACKGROUND: An outbreak of 7 cases (in 6 patients and 1 health care worker, all of whom had AIDS) of multidrug-resistant tuberculosis occurred in a hospital in Chicago. The hospital had a respirator-fit testing program but no acid-fast bacilli isolation rooms. OBJECTIVE: To identify risk factors for transmission of *Mycobacterium tuberculosis*. DESIGN: Retrospective cohort study. SETTING: Private hospital. PARTICIPANTS: Patients and health care workers exposed to *M. tuberculosis*. MEASUREMENTS: Analysis of *M. tuberculosis* isolates, tuberculin skin testing, assessment of exposure, and assessment of participant characteristics. RESULTS: All 7 *M. tuberculosis* isolates had matching DNA fingerprints. Of patients exposed to *M. tuberculosis*, those who developed tuberculosis had lower CD4$^+$ T-lymphocyte counts ($p = 0.02$) and were more likely to be ambulatory ($p = 0.03$) than those who did not. Of 74 exposed health care workers, the 11 (15%) who had conversion on tuberculin skin testing were no more likely than those who did not have conversion to report that they always wore a respirator with a high-efficiency particulate air filter. CONCLUSIONS: Transmission of *M. tuberculosis* occurred in a hospital that did not have recommended isolation rooms. A respirator-fit testing program did not protect health care workers in this setting.

Does a Chest X-Ray Alter the Management of New Patients Attending a Geriatric Day Hospital?—Logan JA, Vallance R, Williams BO, Paul H. Health Bull (Edinb) 1997;55(1):52.

OBJECTIVES: Studies have suggested that routine chest x-ray is never indicated but all new attenders at our day hospitals have a chest x-ray carried out. Our aim was to determine if this investigation altered the clinical management of patients and to try to select those patients in whom a chest x-ray is indicated. DESIGN: A prospective study was carried out over a 7-month period

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from February to September 1995. All new patients had cardiorespiratory symptoms/signs documented and a management plan made. A chest x-ray was then carried out and change in management as a result of the x-ray report noted. SETTING: Knightswood and Drumchapel Geriatric Day Hospitals, West Glasgow University NHS Trust. SUBJECTS: All new Day Hospital attenders. RESULTS: Of 207 new Day Hospital attenders, 53 had no clinical indication for a chest x-ray and although 70% had an abnormal film in no case was patient management changed as a result of this. A chest x-ray was indicated in 154 patients and of these 114 (74%) had an abnormal film with a resultant change in management in 23 patients (this comprised either a change in drug treatment or a further investigation.) Of those whose management was changed as a result of the x-ray report 61% had respiratory symptoms. CONCLUSION: We would recommend that chest x-ray should be performed in those patients who have a clear clinical indication and that the diagnostic yield is highest in the presence of respiratory symptoms or signs.

Reactive Airways Dysfunction Syndrome Due To Chlorine: Sequential Bronchial Biopsies and Functional Assessment—Lemiere C, Malo JL, Boutet M. Eur Respir J 1997;10(1):241.

Very little information is available on the acute histopathological bronchial alterations caused by reactive airways dysfunction syndrome (RADS). We had the opportunity to carry out sequential bronchial biopsies in a subject with RADS due to chlorine (60 h, 15 d, 2 and 5 mo after the acute exposure), and also to assess spirometry and bronchial responsiveness to methacholine. A 36- year-old worker in a water-filtration plant (nonsmoker) abruptly inhaled high concentrations of chlorine on September 12, 1994. He experienced immediate nasal and throat burning, retrosternal burning and wheezing, and these symptoms persisted during and after the workshift. Two days later, he complained of retrosternal burning, dyspnoea and wheezing. Inspiratory wheezing was documented. His forced expiratory volume in 1 second (FEV_1) was 66% of predicted and the provocative concentration of methacholine causing a 20% fall in FEV_1 (PC20) was slightly abnormal (2.5 mg · mL^{-1}). On the following day, the patient underwent bronchial biopsies, which showed almost complete replacement of the epithelium by a fibrinohaemorhagic exsudate. The subject was prescribed inhaled steroids. Fifteen days after the accident, the PC20 was improved to 6 mg · mL^{-1}. Bronchial biopsies showed considerable epithelial desquamation with an inflammatory exudate and swelling of the subepithelial space. Five weeks after the accident, the PC20 was normal (57 mg · mL^{-1}). Inhaled steroids were stopped. Two months after the accident, the PC20 deteriorated to 4 mg · mL^{-1}. Biopsies then showed regeneration of the epithelium by basal cells and there was still a pronounced inflammatory infiltrate. Inhaled steroids were restarted. Three and 5 months later, the PC20 was normal (24 mg · mL^{-1}). Bronchial biopsies showed a greatly improved epithelium and reduction of the inflammatory infiltrate. This case report shows that reactive airways dysfunction syndrome can cause acute, marked, though partially reversible, histological abnormalities. Inhaled steroids may modulate changes in bronchial responsiveness in this condition.

Drownings in Mississippi 1992-1994: Frequencies and Distribution—Carithers T, Higginbotham JC. J Miss State Med Assoc 1997; 38(3):83.

This investigation was conducted to determine the mortality rate due to drownings in Mississippi from 1992-1994. Drownings were defined as unintentional deaths from asphyxia while submerged or within 24 hours of submersion. Death certificate data including ICD-9 codes related to drownings were received from the Mississippi State Department of Health. Variables assessed included age, age category, sex, race, death month, death day, place of drowning, and risk factors. Although drownings occurred in ages from 0-91 years, the highest mortality rates were found in the 14-17 year old age category (11.9 deaths/100,000). Asian and Native American populations had the highest drowning mortality rates (23.6 and 22.6/ 100,000) when analyzed by race. However, this may be primarily due to lower populations. Males drowned 5 times more frequently than women. This is felt to be related to increased exposure. More drownings occurred on Saturdays and Sundays; and in the months of July, June, May, April, September, and August, consecutively. Results presented here identify correlates of drowning fatalities which will enable strategic targeting of prevention programs and resources.

A National Census of Those Attending UK Accident and Emergency Departments with Asthma. The UK National Asthma Task Force—Partridge MR, Latouche D, Trako E, Thurston JG. J Accid Emerg Med 1997;14(1):16.

OBJECTIVE: To obtain a representative national picture of the type of people with asthma attending accident and emergency (A&E) departments in the UK, the reasons why they attend, and to determine the proportion admitted to hospital. DESIGN: A national census involving questionnaires. SETTING: 100 A&E departments throughout the UK. SUBJECTS: All those with asthma attending because of asthma during a 1-week period in September 1994. RESULTS: Details were obtained about 1292 attendances. About half of all attendances were by adults and half by children, and 87.8% were previously diagnosed asthmatics; 18.8% of adult attenders were unemployed. Perceived severity of asthma was the reason for attendance in 65.5%, but 11.5% reported nonavailability, or perceived nonavailability, of the general practitioner (GP) as the reason for attending. One fifth of adults had been kept awake by their asthma for over 3 nights before attendance. Four-hundred and twenty-five of the 1292 attenders (32.9%) had been admitted to hospital in the previous 12 months, and 316 (24.5%) had attended the A&E department in the previous 3 months. Only 24.6% of attenders had had contact with their general practitioner in the previous 24 hours. 61.6% of under-5 attenders (n = 341) were admitted to hospital; the figures for those aged 5-15 and 15+ years and above were 265 (41.4%) and 665 (38.7%). CONCLUSIONS: Many people with asthma attend A&E departments without first having seen their GP. In many adult cases the asthma, while severe, is not acute, but a high proportion of both adults and children are admitted to hospital. Many of these attendances and admissions are repeat attendances. To enhance the quality of care provided to those with asthma may require easier access to primary care, enhanced patient education, or enhanced health professional education. Further study is needed of a variety of potential interventions.

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Airway Pressures and Volutrauma: Is Measuring Tracheal Pressure Worth the Hassle?

Monitoring airway pressures during mechanical ventilation is a standard of care.[1] Sequential recording of airway pressures not only provides information regarding changes in pulmonary impedance but also allows safety parameters to be set. Safety parameters include high- and low-pressure alarms during positive pressure breaths and disconnect alarms. These standards are, of course, based on our experience with volume control ventilation in adults. During pressure control ventilation, monitoring airway pressures remains important, but volume monitoring and alarms are also required.

Airway pressures and work of breathing are also important components of derived variables, including airway resistance, static compliance, dynamic compliance, and intrinsic positive end-expiratory pressure (auto-PEEP), measured at the bedside.[2] The requisite pressures for these variables include peak inspiratory pressure, inspiratory plateau pressure, expiratory plateau pressure, and change in airway pressure within a breath. Plateau pressures should be measured at periods of zero flow during both volume control and pressure control ventilation. Change in airway pressure should be measured relative to change in volume delivery to the lung (pressure-volume loop) to elucidate work of breathing.

SEE THE RELATED STUDY ON PAGE 1179.

Evidence that mechanical ventilation can cause and exacerbate acute lung injury has been steadily mounting.[3-5] While most of this evidence has originated from laboratory animal studies, recent clinical reports appear to support this concept.[6,7] Traditionally, ventilator-induced lung injury brings to mind the clinical picture of tension pneumothorax. Barotrauma (from the root word *baro,* which means pressure) is typically associated with excessive airway pressures. Current thinking regarding ventilator-induced lung injury includes barotrauma, but the real emphasis has shifted to *volutrauma.*

For distinction, barotrauma is extra-alveolar air and includes pneumothorax, pneumomediastinum, pneumoperitoneum, and subcutaneous emphysema. Volutrauma, on the other hand, is lung injury secondary to overdistention of alveoli. Volutrauma is manifested by disruption of the alveolar capillary membrane and alterations in gas exchange that mimic acute lung injury. These distinctions are important because it is not peak airway pressure that is deleterious to the pulmonary architecture. For instance, in a classic example, we suppose that the lung is placed in a rigid container; under this condition, the airway pressures could rise to astronomical values with little to no effect on the pulmonary parenchyma thereby protecting the lung from injury. This protection occurs because the container limits lung inflation. The term volutrauma, then, may be a more representative term for the iatrogenic lung injury seen during mechanical ventilation.

Large tidal volumes are not inherently dangerous; in fact, volutrauma can occur at relatively modest tidal volumes. Volutrauma occurs when individual alveolar units are overstretched. The propensity for development of volutrauma is, therefore, related to the volume of lung available and to regional differences in compliance. In acute respiratory distress syndrome (ARDS) where lung volume is significantly diminished, compliance is reduced, and heterogeneous lung injury is the rule, the risk of volutrauma is magnified. In a postoperative patient with reduced lung volumes and relatively normal compliance, volutrauma is less likely, even at relatively large tidal volumes. This is a quandary that makes setting safe limits on tidal volume for a wide variety of patients extremely difficult.

So where does this leave us? Several groups have espoused the use of small tidal volumes (4-8 mL/kg), pressure-limited ventilation, and permissive hypercarbia.[8,9] Guidelines for maximum airway pressure are based on laboratory data, and typically a plateau pressure < 35 cm H_2O is thought to limit volutrauma.[8,9] In this issue of the Journal, Jager and Tweeddale[10] suggest that monitoring tracheal pressure is a better estimate of volutrauma and barotrauma risk. Their study demonstrates that the presence of the endotracheal tube causes the ventilator to overestimate true peak inspiratory pressure. The pressure drop across endotracheal tubes has been well described, and previous authors have made similar conclusions.[11,12] Jager and Tweeddale suggest, if one is to insist on following peak airway pressure, tracheal airway pressure is the more appropriate measurement.

In several respects, I believe Jager and Tweeddale are correct. Proximal and ventilator airway pressures overestimate the actual airway pressure applied to the lung. Tracheal pressure allows a more accurate estimate of airway resistance

because it eliminates the endotracheal tube as a confounding factor. They also suggest that predicting tracheal pressure from proximal pressure is unreliable. Other authors have agreed and disagreed with this conclusion,[13,14] but this finding has implications for manufacturers who might use estimated tracheal airway pressure to automatically compensate for tracheal tube resistance. Jager and Tweeddale's[10] finding that the ventilator pressure is much higher than a separate measurement of proximal airway pressure is interesting. It is unfortunate that the type of ventilator(s) used in this study[10] is not mentioned. The question then becomes, Is measuring tracheal pressure worth the hassle?

One limitation of this paper[10] is the tracheal pressure measurement technique. When a catheter is placed through the length of the endotracheal tube, the resistance characteristics of that tube are changed. The catheter effectively reduces the diameter of the tube and promotes turbulence. The fact that an endotracheal tube in vivo with a catheter running its length results in poor estimation of tracheal pressure based on in vitro constants measured without the catheter in place is not surprising.

With regard to the utility of proximal airway pressure as currently measured or tracheal airway pressure as suggested by Jager and Tweeddale[10] in establishing risk for volutrauma, I believe the answer is that neither is particularly helpful. The best estimate of alveolar distending pressure is plateau pressure.[2] When airway flow is zero at end-inspiration or end-expiration the plateau pressure is equivalent throughout the respiratory tract. In fact, the use of static pressure-volume curves has been shown to be helpful in identifying both overdistention (the upper inflection point) and optimal PEEP (the lower inflection point) during ventilatory support of patients with acute lung injury.[7,8] Therefore, I believe it is not only practical but also prudent to continue to measure peak inspiratory pressure during routine ventilator care. This pressure is readily available, has been used successfully for decades, and provides the kind of information required for routine monitoring. Jager and Tweeddale[10] are correct, peak inspiratory pressure is a poor predictor of alveolar pressure and can be misleading during the comparison of ventilatory modalities. But the measurement of tracheal pressure only adds complexity. In the face of rising airway pressure and concern for the development of ventilator-induced lung injury, plateau pressures should be used to estimate alveolar pressure, and static pressure-volume curves should be used to evaluate tidal volume and PEEP settings.

Another important point should be made regarding the example of the lung in a rigid container. The lung is normally in a semirigid container—the chest wall. Under this condition, the transalveolar pressure changes with changes in chest-wall compliance. This has clinical significance. The elderly patient with pneumonia and chronic lung disease and with mechanical ventilation for acute lung injury has an entirely different tolerance for airway pressures than the young trauma patient with acute lung injury following massive blood and fluid resuscitation. In the first case, the patient with relatively normal chest-wall compliance may only tolerate 30 cm H_2O of plateau pressure before regional alveolar overdistention occurs. In the second case, higher airway pressures may be tolerated due to the relatively stiff chest wall. The risk of volutrauma then, is best judged by transalveolar pressure (alveolar pressure-pleural pressure). This value does not easily lend itself to routine measurement, but the use of static pressure-volume curves does appear to offer some assistance in this area.

Jager and Tweeddale[10] have provided us with further evidence that plateau pressure is the key to monitoring alveolar pressure. At present, static pressure-volume curves require time and expertise to accomplish correctly. However, a recent report has demonstrated that under appropriate conditions, upper and lower inflection points can be obtained during a single, slow passive inflation.[15] This technology may become extremely useful as we continue to care for patients with ARDS into the year 2000.

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Comparison of Circulaire™ to Conventional Small Volume Nebulizer for the Treatment of Bronchospasm in the Emergency Department

Lee Hoffman MD and Howard Smithline MD

INTRODUCTION: The Circulaire™ (C-Neb), a hand-held small volume nebulizer, has been previously shown to have superior performance characteristics: greater peripheral lung deposition, reduced gastrointestinal system deposition, and reduced aerosol loss to the environment. This study evaluated whether the increased lung deposition would yield an improvement in pulmonary function without increasing extrapulmonary side effects. METHODS: All patients entering our Emergency Department with acute bronchospasm requiring aerosolized β-agonist therapy were randomized to either the C-Neb or the hospital's standard nebulizer. Patients were blinded to the purpose of the study. RESULTS: At 20 minutes post-treatment the C-Neb had a greater improvement in peak expiratory flow, heart rate, and respiratory rate compared to the standard nebulizer. There was no difference in blood pressure, tremulousness, and nausea. The C-Neb required less time to complete the aerosolization. CONCLUSION: The C-Neb had a superior clinical profile compared to the standard nebulizer for the initial treatment of patients with acute bronchospasm. [Respir Care 1997; 42(12):1170-1174]

Introduction

Inhalation therapy is the mainstay of asthma treatment. In theory, aerosolized β agonists are effective bronchodilators with limited extrapulmonary side effects because of reduced systemic absorption compared to oral or subcutaneous β agonists.[1] It is unfortunate that all the devices currently available for generating respirable aerosols are inefficient and deliver only 5 to 10% of the medication initially placed in the devices to the lungs.[2,3] The remainder of the medication is absorbed through the gastrointestinal (GI) tract or vented into the atmosphere.

The nebulizer, Circulaire™ (C-Neb),* is currently being marketed as being superior to other nebulizers because it produces smaller and more uniform particles. More medication is deposited in the lower airways and less is deposited in the pharynx, mouth, and stomach.[4] This may lead to improved pulmonary response and decreased side effects with each aerosolized treatment; however, this has never been demonstrated in acute asthmatics. Additionally, the device is designed to deliver the medication in 2-4 minutes compared to 10-15 minutes for the standard nebulizer.[5] This also has never been demonstrated in a clinical setting.

We hypothesized that acute asthmatics treated with β agonists will have a greater pulmonary response and decreased side effects when the β agonist is aerosolized by the Circulaire compared to our hospital's standard nebulizer. We also hypothesized that the time to deliver this medication would be reduced.

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The authors have no financial interest in any of the products mentioned in this paper.

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*Suppliers of commercial products are listed in the Product Sources section following the text.

Future studies will be needed to evaluate whether use of the C-Neb affects outcome variables such as rate of hospitalization, duration of acute treatment, time to recovery, and cost-benefit ratios. The topic of aerosolization of medication has previously been extensively reviewed.[6-9]

Methods

The study was approved, and informed consent was waived by the hospital's Institutional Review Board.

Nebulizers

The two devices studied were the Airlife™ Misty-Neb™ (or M-Neb, the standard small volume hand-held nebulizer used in our institution) and the Circulaire (Fig. 1). The Circulaire has three principle differences. It uses a VixOne™ aerosol generator which has been shown to have superior performance characteristics,[5] a reservoir bag which traps large diameter particles, and a variable resistor which allows for positive end-expiratory pressure within the system which may have therapeutic benefit.[10]

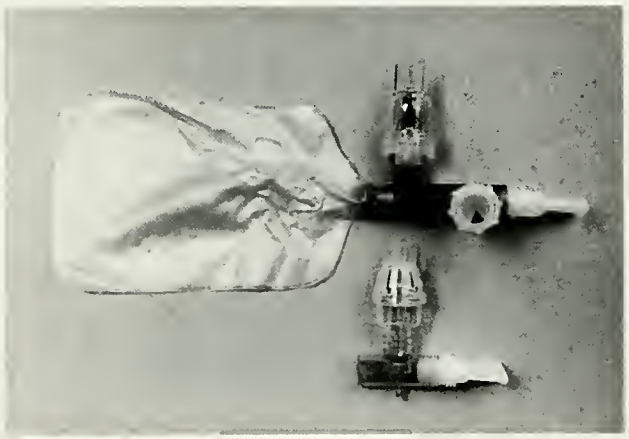

Fig. 1. The Circulaire™ (top) and the Airlife™ Misty-Neb™ small volume nebulizers.

Each aerosolized β-agonist treatment consisted of nebulizing 0.5 mL of albuterol using an oxygen flow of 8 L/min. The albuterol was diluted with 3-mL saline for the M-Neb, while only 1.5 mL of saline were used for the C-Neb. The amount of diluent was based on the manufacturers' recommendations.

Study Design & Population

Every patient presenting to the Emergency Department (ED) between December 1995 and February 1996 and requiring aerosolized β-agonist treatment was prospectively randomized to receive the medication either by the M-Neb or the C-Neb. Patients were excluded from the study if they were incapable of holding the nebulizer or of performing a forced exhalation to measure a peak expiratory flow (PEFR). Patients were not excluded if they had received a prior aerosolized β-agonist treatment prehospital or in the ED. However, patients must not have received an aerosolized β-agonist treatment within 30 minutes of beginning the study.

Randomization was predetermined with a published random number table and a blocking factor of 4. The data sheets and group allocation were stored in sequentially numbered sealed study packets. The patients were blinded to the nature of the study. It was not possible to blind the respiratory therapists or nurses.

Patients requiring multiple treatments used the same nebulizer for each treatment. Patients were dropped from subsequent data collection if an aerosolized medication other than albuterol was given or if a larger amount of albuterol was given to the patient. At our institution 1.5 mL of albuterol diluted to 15 mL is sometimes given to patients continuously over one hour instead of three intermittent 0.5-mL treatments. Patients who received this continuous treatment were dropped from subsequent data collection.

Oral or intravenous steroids were given at the discretion of the treating physician. The patient was excluded if steroids had been given prior to entry into the study.

Measurements

Pretreatment and 20-minute post-treatment data were collected for each nebulized treatment. The efficacy of the nebulizers was determined by measuring the respiratory rate (RR) and the PEFR. PEFR was measured using one Assess™ Peak Flow Meter. Patients were instructed in the use of the peak flow meter by a respiratory therapist or nurse. The highest measurement out of 3 attempts was recorded.

Side effects were measured by recording the change in the degree of nausea and tremulousness. Patients reported their responses on a 3-point scale (increased, unchanged, decreased). Heart rate (HR), systolic blood pressure (SBP), and diastolic blood pressure (DBP) were also recorded as measures of both efficacy and side effects of the treatments. The treatment duration was recorded. This was the time interval from when the airflow started to when no further aerosolization could be seen.

Statistical Analysis

Pretreatment, post-treatment, and absolute change in PEFR, HR, RR, SBP, DBP, and the treatment duration were analyzed using the Mann-Whitney U—Wilcoxon Rank Sum Test. Fisher's Exact Test was used to analyze the change in nausea and tremulousness. Data from a patient's first aerosolized treatment were analyzed separately from data of subsequent treatments because of significant patient attrition. All analyses were 2-tailed, and a p value < 0.05 was considered statistically sig-

nificant. The commercial computer program SPSS Version 6.1 was used for statistical analysis. Enrollment was set at 150 patients to ensure complete data sets for at least 65 patients in each group. This was calculated to yield a power of 0.95 to detect a 40 L/min change in PEFR.

Results

One-hundred fifty patients were enrolled. Five patients in the M-Neb group and 11 in the C-Neb group were dropped from analysis because post-treatment data were not recorded. All results were, therefore, based on 70 patients in the M-Neb group and 64 patients in the C-Neb group.

The baseline (pretreatment) characteristics of the 2 groups were similar except for the DBP which was statistically different but probably of no clinical significance (Table 1).

Table 1. Baseline, or Pretreatment, Mean Values for the 2 Study Groups.

	Airlife™ Misty-Neb™ (N = 70)	Circulaire™ (N = 64)	p Value
Age (years)	43	48	0.218
Male (%)*	57	52	0.603
Prev tx (%)*	26	20	0.540
PEFR (L/min)	170	190	0.713
RR (min-1)	24	24	0.640
HR (min-1)	100	98	0.957
SBP (mm Hg)	127	130	0.081
DBP (mm Hg)	77	80	0.023

*Values are percentages.
Prev tx = the percent of patients receiving an albuterol treatment prior to entry into the study; PEFR = peak expiratory flow; RR = respiratory rate; HR = heart rate; SBP = systolic blood pressure; DBP = diastolic blood pressure.

The C-Neb yielded a statistically significant greater PEFR compared to the M-Neb after the first aerosolized treatment, $p < 0.001$. The median absolute change in PEFR for the M-Neb Group was 30 L/min, while the median change in the C-Neb Group was 80 L/min (Fig. 2). Frequency distributions of the change in PEFR for each group approximates a normal distribution. Five C-Neb patients had extremely high changes in their PEFR and may represent outliers (Fig. 3); however, post-hoc analysis excluding these patients did not alter our results. There was no significant difference in post-treatment HR, RR, or SBP. The absolute change in HR and RR was significantly different for the 2 groups, but these differences (0 vs 5 beats/min for C-Neb and 2 vs 2 breaths/min for M-Neb) may be of little clinical significance. The post-treatment diastolic blood pressure was statistically different between the 2 groups, but this difference (74 mm Hg for C-Neb vs 80 mm Hg for M-Neb) also is probably of no clinical significance. Additionally, the treatment duration was significantly less in the C-Neb Group compared to the M-Neb Group, $p < 0.001$. The median time durations were 5 minutes for the C-Neb Group and 12 minutes for the M-Neb Group.

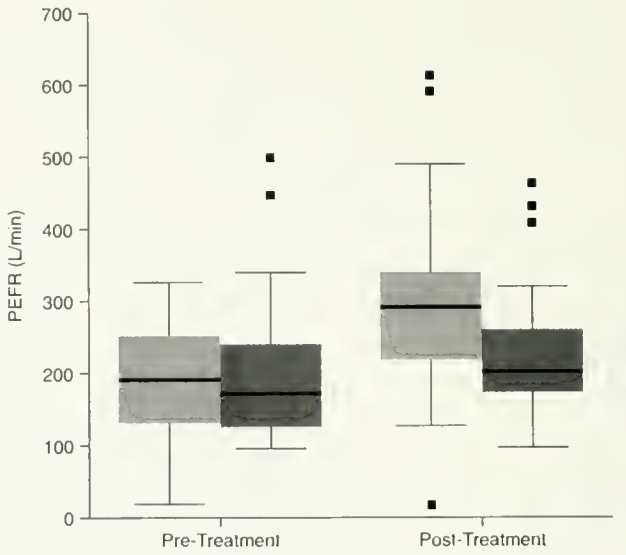

Fig. 2. Comparison of peak expiratory flow for both groups before and after the first treatment with albuterol. The lower boundary of each box is the 25th percentile, the upper boundary is the 75th percentile. The horizontal in inside each box represents the median or 50th percentile. Outlier values > 1.5 box lengths from the lower or upper edge of each box are represented by a solid square. The whiskers connect the largest and smallest nonoutlier values. ■ = C-Neb; ■ = M-Neb.

Table 2. The Number of Patients Reporting a Change in Nausea and Tremulousness with the First Treatment of Albuterol.*

	Nausea	
	Airlife™ Misty-Neb™	Circulaire™
Increase	2	2
No change†	65	62
	Tremulousness	
	Airlife™ Misty-Neb™	Circulaire™
Increase	25	15
No change	42	49

*There was no significant difference between the 2 study groups.
†No change also includes the number of patients reporting a decrease in symptoms.

The nausea and tremulousness data were collapsed into 2×2 tables. Fisher's Exact Test showed no statistical difference between the 2 groups, $p > 0.999$ for nausea, and $p = 0.092$ for tremulousness (Table 2).

Data from a patient's second treatment were sparse because of protocol violations (primarily because of using a 1-hour continuous treatment with 1.5-mL albuterol). There were 42 patients (22 M-Neb, 20 C-Neb) who received a second treat-

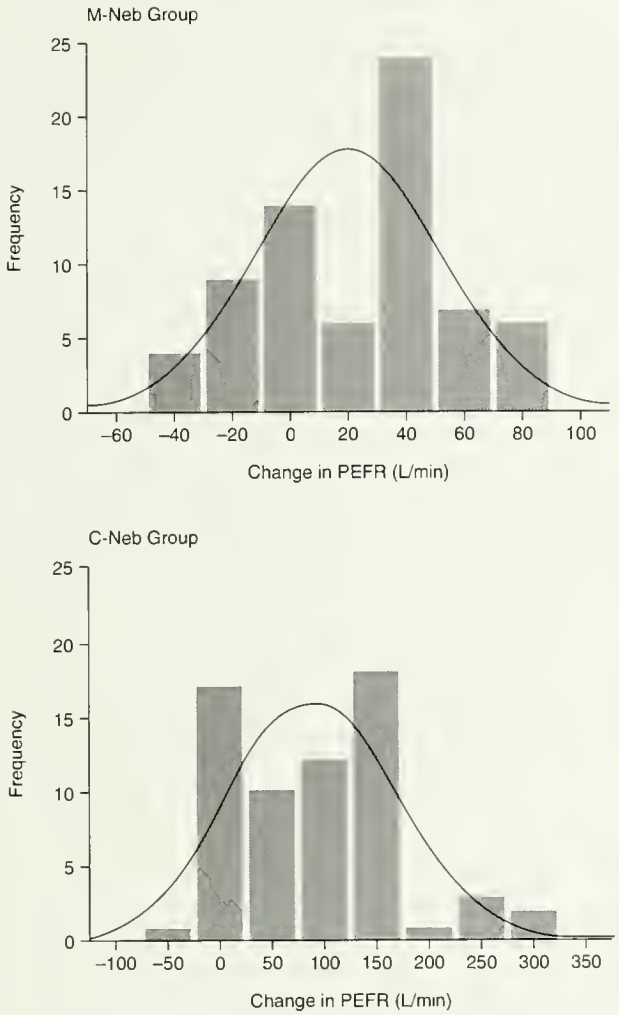

Fig. 3. The histograms record the frequency of change in peak expiratory flow (PEFR) for the 2 groups. The frequency is superimposed over the normal distribution. The 2 bars centered around 250 and 300 L/min for the Circulaire Group may represent 5 outliers.

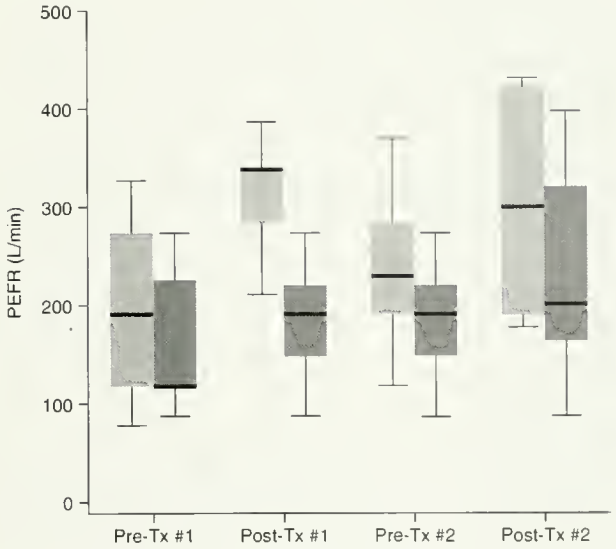

Fig. 4. Comparison of peak expiratory flow (PEFR) in both groups with measurements over 2 treatments. The box plots represent the first and second treatments. The Circulaire (C-Neb) Group is statistically greater than the Misty-Neb (M-Neb) Group posttreatment 1 and 2. However, there was no statistical difference between posttreatments 1 and 2 for the C-Neb. ▒ = C-Neb; ■ = M-Neb.

ment without breaking protocol. Overall the C-Neb group had a significantly greater PEFR than the M-Neb group after both the first treatment and second treatment ($p > 0.05$); however, most of the improvement with the C-Neb in the first treatment had dissipated by the time the second treatment had started (Fig. 4). The duration between treatment was the same for both groups. There was no statistically significant increase in PEFR comparing the post-treatment data between Treatments 1 and 2 for C-Neb ($p = 0.570$, Wilcoxon Matched-Pairs Signed-Ranks Test); however it was significant for M-Neb ($p = 0.008$, Wilcoxon Matched-Pairs Signed-Ranks Test).

Discussion

We have shown that under the conditions of this study, acute asthmatics get a better bronchodilator response from the C-Neb than from the M-Neb as demonstrated by improvement in PEFR. A further indicator that breathing was eased with the C-Neb is the greater decrease in respiratory rate. Also of interest is the decrease in heart rate seen in the C-Neb treatment group. This may reflect more relaxed breathing and consequently less anxiety. The preferential decrease in heart rate with C-Neb may also reflect less systemic absorption of the bronchodilator with less sympathetic stimulation of the heart despite a greater amount of the medication being delivered to the lungs. This probably occurs because less medication is impacting the oropharynx and being absorbed by the gastrointestinal system.[4]

Consistent with this interpretation is the additional finding that fewer patients treated with C-Neb experienced tremulousness, a well recognized untoward effect related to systemic absorption of sympathomimetic drugs.

It is difficult to conclude much from our data concerning subsequent treatments because of the small sample size. The lack of improvement between the post-treatment PEFR data and between the first and second treatments for the C-Neb Group suggests that the maximal β-receptor stimulation may have been reached after the first treatment. However, because of the small number of patients, there was only sufficient statistical power to detect a 75 L/min difference.

These 2 nebulizers have already been compared by Mason and colleagues (1994),[4] and Mason and Miller (1996).[11] In the first article 9 normal subjects inhaled 99mTc-diethylenetriamine pentaacetate aerosolized by the C-Neb and the M-Neb. Aerosol escaping from the nebulizer during exhalation

was trapped and counted. Extrapulmonary deposition of counts was estimated. They[4] concluded that the C-Neb produced a statistically significant improvement in lung deposition, a reduction in extrapulmonary deposition, and a reduction in the amount of aerosol escaping to the environment. Our results are concordant with their findings. The second study[11] compared C-Neb to M-Neb in 10 patients with stable COPD. However unlike our study, pulmonary function (as measured by forced expiratory volume in the first second, 5 to 10 minutes post-treatment) was the same for both nebulizers. Additionally, contrary to their first study, they did not show an improved penetration of isotope-labeled aerosol into the periphery of the lung with C-Neb. However, there was significantly less extrapulmonary deposition. The lack of improvement in airflow may be because they evaluated 'stable' COPD patients who had less intrinsic airway hypersensitivity and/or they may not have allowed sufficient time for the albuterol to work.

Our study was limited by the evaluation of patient response to, at most, only 2 treatments and by not clearly treating patients to maximal β-receptor stimulation. We also did not compare patient height for the 2 groups which may have an impact on interpreting their severity of disease. However, there is no reason to suspect a nonrandom distribution between the 2 groups. Our design was necessary to avoid interference with routine patient care. It is also unclear how much time is actually saved by using the Circulaire. In light of our results, additional studies evaluating outcome measures such as admission rates, time to improvement, time to maximal β-receptor stimulation, and cost need to be completed.

In summary, we tested the C-Neb by comparing it with a conventional small volume nebulizer on unselected patients thought to have acute bronchospasm by clinical examination in a busy Emergency Department. We found the C-Neb to be superior in terms of improvement of airflow without an increase in side effects. Treatment time was decreased by giving C-Neb treatments with twice the usual concentration of medication.

PRODUCT SOURCES

Circulaire™, WestMed Inc, Tuscon AZ
Airlife™ Misty-Neb™, Baxter HealthCare Corp, Valencia CA
Assess™ Peak Flow Meter, Healthscan Products Inc, Cedar Grove NJ
SPSS™ version 6.1, SPSS Inc, Chicago IL

ACKNOWLEDGMENTS

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The Effect of Plasmapheresis on Respiratory Muscle Strength in Patients with Guillain-Barré Syndrome

Jonathan E Kass MD, David Gerber DO, Ritha Kartan MD, Thaddeus Bartter MD, and Melvin R Pratter MD

STUDY OBJECTIVES: To evaluate the effect of plasmapheresis on respiratory muscle function in patients with Guillain-Barré Syndrome (GBS). DESIGN: A prospective study. SETTING: A university hospital. PATIENTS: Fifteen consecutive patients with GBS. MEASUREMENTS: Vital capacity (VC) and negative inspiratory force (NIF) immediately prior to and 6, 12, and 24 hours after each plasmapheresis treatment. RESULTS: For each plasmapheresis session, there was no significant difference ($p > 0.1$) between the baseline (preplasmapheresis) and any of the 6-, 12-, or 24-hour postplasmapheresis VC measurements. When serial baseline measurements of VC were compared, there were significant differences between the baseline value (standard deviation, SD) for Session 1 [1.61 (0.95) L] and baseline values (SD) for Session 4 [1.89 (1.1) L, $p < 0.05$], Session 5 [1.88 (1.1) L, $p < 0.01$], and Session 6 [1.89 (1.2) L, $p < 0.01$]. As with the VC, the baseline NIF did not differ significantly from the acute postplasmapheresis values for any session. When the serial baseline NIF measurements were compared, the only significant difference ($p < 0.05$) was between Session 1 [46.8 (24.7) cm] and Session 5 [57.5 (23.9) cm]. CONCLUSION: While each individual plasmapheresis has no immediate effect upon respiratory muscle strength (RMS) in patients with GBS, there does appear to be a cumulative beneficial effect with improvement in RMS apparent by the start of the fourth session. However without a randomized controlled trial, spontaneous improvement cannot be excluded as the cause for the changes observed over time. [Respir Care 1997;42(12):1175-1178]

Introduction

The Guillain-Barré Syndrome (GBS) is an acute demyelinating polyneuropathy of presumed immunologic etiology. It usually presents as an ascending motor paralysis and is most often, though not always, seen following an infectious (often viral) illness. Although relatively rare, with an estimated annual incidence of only 1 case/75,000-100,000 people, it remains the most commonly observed acute polyneuropathy.[1] Acute respiratory failure is one of the most common and serious complications of GBS, and the range of reported incidence of acute respiratory failure in GBS is 10 to 80%.[2-7]

Over the last decade, plasmapheresis has become a standard therapy for GBS and has been shown to shorten the course of the disease.[1] Although patients treated with plasmapheresis have been shown to require mechanical ventilation for shorter periods of time than those not receiving plasmapheresis,[8,9] the acute effect of individual plasmapheresis treatments on respiratory muscle strength (RMS) has not been assessed. We

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chose to evaluate the effect of plasmapheresis on standard measures of respiratory muscle function in patients with GBS.

Methods

We prospectively studied 15 consecutive patients with GBS admitted to our institution over a 2-year period (1992-1994), all of whom were treated with plasmapheresis. All patients met the current diagnostic criteria for GBS.[10] At the time of study enrollment, 4 of the patients were being supported with mechanical ventilation, and 11 patients had not developed respiratory failure. There were 9 men and 6 women, with a mean (standard deviation, SD) age of 49.0 (21.2) years. The patients did not have any other known acute or chronic cardiopulmonary disease. The mean time (SD) from diagnosis to the start of plasmapheresis was 1.9 (1.8) days. Because plasmapheresis has been shown to be beneficial in the treatment of GBS,[8,9,11] it was concluded that a nontreatment control group was not ethical.

Each plasmapheresis was performed by removing 4 L of plasma and replacing it with 4 L of purified plasma fraction. According to protocol, the first 2 plasmaphereses were performed 24 hours apart, with subsequent sessions performed every 48 hours thereafter. The goal was for each patient to have plasmapheresis 5-6 times unless complications occurred.

Three baseline measurements of RMS, vital capacity (VC), and negative inspiratory force (NIF) were obtained immediately prior to each plasmapheresis. In order to document any acute effect of plasmapheresis upon RMS, repeat measurements were made 6, 12, and 24 hours after each session. All measurements were made by licensed respiratory therapists using a Ventilator Monitoring Adapter Circuit™ (Baxter Health Care Corporation, Deerfield IL) and a pressure gauge with the patients in the upright position. Patients on mechanical ventilation were taken off of the ventilator for all measurements. Three maximal efforts, as determined by the respiratory therapist, were performed for each measurement, and the value was taken as the best of the 3 efforts. NIF measurements were made with a 1-way valve so that NIF was obtained near residual volume rather than functional residual capacity.

Decisions regarding intubation and extubation were made independently by the patients' critical care attending physicians, who did not have access to all of the RMS measurements.

Statistics

The data are presented as mean (SD). An analysis of variance with repeated measures was used to assess the differences between the multiple measurements of VC and NIF separately.

Results

A total of 83 plasmaphereses were performed during the study period. Fourteen of the 15 patients received 5 plasmaphereses. The mean number of sessions per patient was 5.5. One patient stopped after 3 sessions because of hypotension and urticaria.

The preplasmapheresis baseline measurements are shown in Table 1. Muscle weakness was documented, with the mean VC 38.7 (17.1)% of predicted.

For each plasmapheresis session, there was no significant difference ($p > 0.1$) between the baseline and any of the 6-,

Table 1. Preplasmapheresis Baseline Data

Patient #	VC* (L)	VC % predicted	VC/kg	NIF	Intubated at outset
1	1.40	43	15.8	−48	No
2	0.50	16	8.1	−20	Yes
3	1.30	37	18.7	−20	No
4	4.02	76	55.0	−50	No
5	1.85	37	14.8	−80	No
6	1.25	43	25.0	−36	No
7	2.00	47	22.8	−48	No
8	2.40	43	38.3	−60	No
9	2.40	44	33.6	−80	No
10	1.55	42	21.3	−70	No
11	1.75	44	40.1	−80	No
12	2.10	54	28.0	−60	No
13	0.10	2	1.6	−10	Yes
14	0.70	17.5	7.9	−20	Yes
15	0.90	35	14.8	−20	Yes
Mean (standard deviation)	1.61 (0.95)	38.7 (17.1)	23.1 (14.2)	−46.8 (24.7)	

*VC = vital capacity, NIF = negative inspiratory force.

12-, or 24-hour postplasmapheresis VC measurements. When serial baseline measurements of VC were compared, there were significant differences between baseline Session 1 [1.61 (0.95) L] and baseline Session 4 [1.89 (1.1) L, $p < 0.05$], Session 5 [1.88 (1.1) L, $p < 0.01$], and Session 6 [1.89 (1.2) L, $p < 0.01$] (Fig. 1).

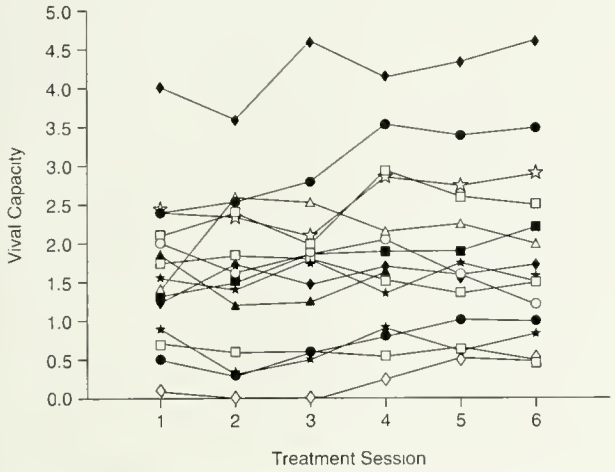

Fig. 1. Vital capacity in relation to treatments (values prior to each treatment). For every patient in the study, vital capacity is charted against treatment session (not study day).

As with the VC, the baseline NIF did not differ significantly from the acute postplasmapheresis values for any session. When the serial baseline NIF measurements were compared, the only significant difference ($p < 0.05$) was between Session 1 [46.8 (24.7) cm] and Session 5 [57.5 (23.9) cm]. To see the response of the patients with the worst initial values, the subset of 5 patients with an NIF of −20 or worse was examined (Fig. 2). Three of the 5 had improved by Session 3. There were too few presession 6 NIF measurements for adequate statistical analysis.

Eleven patients were not on a ventilator at the outset of the study. None of 7 patients who had an improved VC by Day 6 (baseline Session 4) required intubation. In contrast, 2 of the 4 patients who did not improve their VC by Day 6 ultimately required intubation.

Discussion

Our study has documented a slow sequential improvement in respiratory muscle strength despite the lack of acute improvement following serial plasmaphereses in patients with GBS. Measurements of VC and NIF taken at 6, 12, and 24 hours after each session did not differ significantly from the baseline values for that session. The VC was significantly improved by Day 6 (Session 4), and the NIF became significantly better by Day 8 (Session 5). Although the data suggest that plasmapheresis led to a gradual improvement in RMS, a learning effect on pulmonary function performance and/or spontaneous improvement over time cannot be excluded. The lack, for ethical reasons, of an untreated control group prevented us from being able to address the issue of spontaneous improvement versus a plasmapheresis effect.

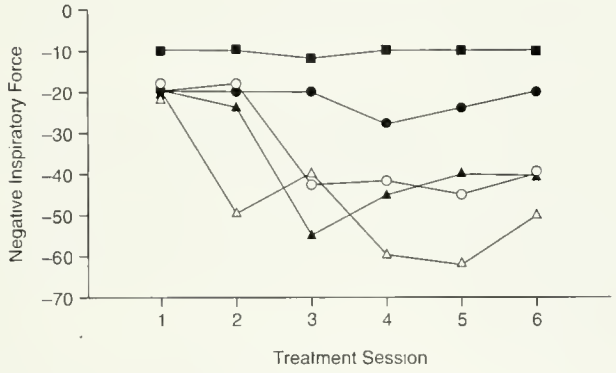

Fig. 2. The subgroup of 5 patients with the lowest baseline negative inspiratory force (NIF). NIF charted against treatment session (not study day).

Previous controlled studies of plasmapheresis in GBS have documented an improvement over time in peripheral muscle strength although none has looked at the acute (immediately postsession) effects of plasmapheresis on peripheral muscle strength.[8,9,11] The literature on RMS is even more sparse. One study has suggested indirectly that plasmapheresis improves RMS:[8] patients with GBS treated with plasmapheresis required shorter periods of mechanical ventilation than patients not receiving plasmapheresis. To our knowledge, no prior study has directly evaluated the acute effects on RMS or any change over time in RMS.

Our study protocol did not include objective measurements of peripheral muscle strength or other neurological findings contemporaneously with RMS. We cannot therefore document the extent to which the cumulative improvement in RMS corresponded with an improvement in peripheral muscle strength. However, it has been shown that assessment of peripheral muscle strength in stable chronic neuromuscular disorders cannot be used to predict respiratory muscle function.[12] Thus a direct measurement of RMS is needed. VC has been shown to be a good indicator of RMS.[13,14] According to Macklem[15], the VC is a much more accurate measurement of RMS than is an indirect assessment by subjective peripheral muscle strength scale. VC has also been shown to be helpful in GBS for clinical transitions, for predicting the need for intubation and mechanical ventilation and for predicting when weaning from mechanical ventilation should be successful.[16]

NIF has been shown to be highly dependent upon technique,[17] whereas VC has excellent reproducibility.[18] This may account for the fact that although in the present study improvement in NIF paralleled the increase in VC, the improvement

for the group as a whole did not reach significance until 2 days after the VC. There was also a large amount of individual variation (Fig. 2).

In conclusion, each individual plasmapheresis had no immediate effect upon RMS in patients with GBS. There was improvement in RMS over time which reached significance by the start of the fourth session. While previous studies of peripheral muscle strength lead us to believe that the plasmaphereses played a role in the improvement in RMS, a randomized controlled trial would be needed to confirm this impression.

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In-Vivo Comparison of Measured Tracheal Pressure and Tracheal Pressure Determined by a Simple Bedside Equation

Kelly Jager RRT and Martin Tweeddale MB

BACKGROUND: High peak airway pressure (P_{aw}) has been associated with ventilator-induced lung injury. However, in intubated patients, a marked pressure drop can occur across the endotracheal tube (ETT) thereby making intratracheal airway pressures (P_{trach}) more accurate than peak P_{aw} as read from the ventilator. We sought to determine whether an equation based on in-vitro values for ETT resistance (R_{ett}) reported by Gottfried et al could be used at the bedside to accurately estimate P_{trach} in ventilated critical care patients. METHOD: P_{trach} was estimated by P_{1rach} = (estimated R_{pt})(flow) + $P_{plateau}$, (where R_{pt} is patient resistance determined as respiratory system resistance minus R_{ett}, and $P_{plateau}$ is the pressure read from the ventilator manometer after a period of no flow). Estimated P_{trach} was compared to measured P_{trach} obtained by using a small-bore catheter placed distal to the tip of the ETT. In 10 patients (Group 1), ventilator peak P_{aw}, true proximal peak P_{aw}, P_{trach}, and $P_{plateau}$ were measured and averaged over 3 breaths at the patient's set flow. In a second set of 10 patients (Group 2), the same measurements were made at 4 different flows. In both groups bias ± precision was used to compare estimated P_{trach} and measured P_{trach}. We defined limits of agreement of ± 3.0 cm H_2O as clinically acceptable. In Group 2 patients, ANOVA was used to compare bias and precision of P_{1rach} at 4 different flows. $p < 0.05$ was considered statistically significant. RESULTS: In Group 1, bias ± precision of P_{trach} was −8.8 ± 6.87 cm H_2O when ventilator peak P_{aw} was substituted in the formula but only −2.0 ± 3.5 cm H_2O when proximal peak P_{aw} was used. In Group 2 patients when ventilator peak P_{aw} was used, bias ± precision increased with increasing flows, with bias ± precision at 120 L/min (−10.2 ± 3.83) significantly different from bias ± precision at 60 L (−1.3 ± 1.58) and 80 L/min (−5.0 ± 0.71). CONCLUSIONS: Estimates of peak tracheal pressure based on in-vitro determinations of endotracheal tube resistance may not accurately reflect directly measured values for tracheal pressure. [Respir Care 1997; 42(12):1179-1183]

Introduction

Elevated peak airway pressures (P_{aw}) have been associated with ventilator-associated lung injury,[1-3] and it is presumably for this reason that many practitioners choose to limit peak P_{aw} to approximately 50 cm H_2O during mechanical ventilation (even when plateau pressures, or maximum pressures at points of no flow, are < 35-40 cm H_2O[4]). In intubated patients, peak P_{aw} is the sum of pressures required to overcome the resistance and compliance of ventilator circuitry (which may vary depending upon where the measurement is taken), endotracheal tube (ETT), airways, and lungs: there-

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fore, peak P_{aw} is not indicative of the pressure imposed on patient airways. It is important to remember that a marked pressure 'drop' can occur across the ETT. For example, using constants derived from in-vitro testing,[5] the pressure drop across an ETT with an inside diameter (ID) of 7.0 mm at an inspiratory flow of 120 L/min is 34 cm H_2O (Fig. 1). For this reason, it has been recommended that P_{aw} measurements be made distal to the ETT (P_{trach}) to eliminate that component of total peak P_{aw} due to ETT resistance.[6,7]

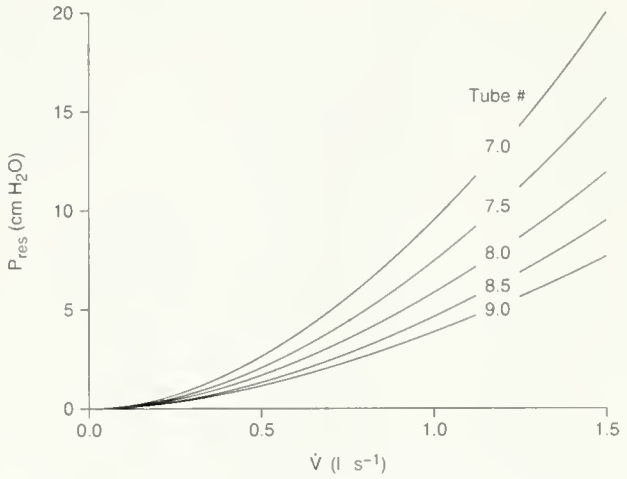

Fig. 1. The relationship between resistive pressure (P_{res}) and air flow ($\dot{V}$) for endotracheal tubes (and connectors) of representative sizes is shown. Also indicated are the results obtained by fitting measurements to a power function of the form $P_{res} = a\dot{V}^b$, where a and b are constants (r = correlation coefficient). (Reprinted from Reference 5, with permission.)

P_{trach} may be directly measured or estimated from in-vitro constants for R_{ett}. P_{trach} can be directly measured by introducing a small-bore catheter through the ETT into the trachea. The procedure is relatively simple but requires time and additional equipment and presents the potential disadvantage of additional airway resistance caused by the presence of the catheter or even total catheter obstruction and malfunction due to plugging with secretions. We sought to determine whether a simple equation based on published R_{ett} values could be used at the bedside to accurately estimate P_{trach} in ventilated critical care patients.

SEE THE RELATED EDITORIAL ON PAGE 1167.

Methods

Catheter Validation

Prior to the clinical study, an in-vitro validation was done of the improvised, recycled, air-filled, end-hole catheters used to measure P_{trach}. The catheters were made from sterilized outer sheaths of used cytology brushes.* Two catheters can be made from each sheath. Three different catheters were studied under two different test conditions. For the first, the test lung had a set compliance of 50 mL/cm H_2O, and the only added resistance was an ETT with an 8.5-mm ID. In the second test condition, 2 parabolic resistors with an inspiratory resistance of 24.5 cm $H_2O \cdot L \cdot s^{-1}$ at a flow of 30 L/min (one in each 'bronchus') added resistance in order to achieve a wider range of pressure measurements in the test lung. Comparisons were made at flows of 60, 80, 100, and 120 L/min. Each of 3 catheters was inserted, one at a time, to 0.5-1.0 cm beyond the end of the ETT into the test lung and connected to a transducer. For this validation, reference lateral pressure was measured directly from a transducer-equipped sidearm in the test lung located just proximal to the 'carina.' Both pressure signals were digitized by a computer equipped with a data-acquisition system. Simultaneous lateral and catheter P_{aw}s (averaged over 3 consecutive breaths) were then recorded for each of the 3 catheters at each ventilator setting.

Patient Studies

In many patients, the apparatus for measuring P_{trach} was already in place to assist us with patient-ventilator care. If not already in place, the catheter was measured by first noting the cut length of the ETT, adding 7.5 cm for the ETT connector and jet adapter, then adding 0.5 cm and marking the catheter at this length. After the standard adapter was replaced by a jet adapter (Fig. 2), one end of the catheter was inserted into the sealed hub of the jet adapter and left in this position until measurements of P_{trach} were needed. The other end of the catheter was then connected to a calibrated pressure transducer.

Patient Investigation: Part 1

Patients were entered into the study if they were ventilated in the assist/control mode with a square inspiratory flow waveform and were not assisting above the set rate. Because experimental conditions did not deviate from the standard of care, institutional review board permission was not necessary. All patients were studied in the supine position, and no ventilator settings were changed during the measurement period. Pressure measurements were taken both from the ventilator and from the catheter-transducer system. First, 3 peak P_{aw} measurements were recorded from the ventilator, and the mean value calculated and recorded. Second, 3 peak P_{aw} measurements were recorded from the catheter-transducer system, with the catheter sitting in the ETT adapter hub (prox-

*Suppliers of commercial products are listed in the Product Sources section following the text.

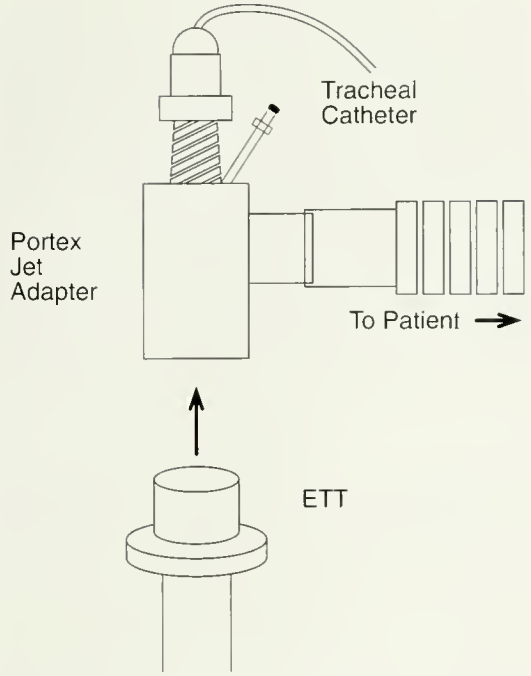

Fig. 2. Schema of test setup. ETT = endotracheal tube.

imal pressure). The mean value was calculated and recorded. Third, the catheter was advanced up to its marked length, which placed the catheter tip 0.5-1.0 cm beyond the end of the ETT. P_{trach} was then recorded from the catheter-transducer system, and the mean value calculated and recorded (distal pressure). Three plateau pressures, 2 seconds in length (or as long as possible), were then recorded from the catheter-transducer system, and the mean value calculated and recorded.

Patient Investigation: Part 2

A second group of patients was studied using the same protocol with the exception that all measurements were repeated at each of 4 flows (60, 80, 100, and 120 L/min) in each patient.

Measurements & Calculations

Values were recorded for ETT ID and length, duration of intubation, flow, tidal volume, total positive end-expiratory pressure (PEEP), and patient diagnosis. Values were calculated for respiratory system compliance (C_{rs}) and total inspiratory respiratory system resistance (R_{rs}):

$$C_{rs} = \frac{\text{effective tidal volume}}{\text{plateau} - PEEP_{tot}}, \text{ and}$$

$$R_{rs} = \frac{P_{aw} - P_{plateau}}{\text{flow}}. \quad [1]$$

The measured R_{pt} ($R_{pt\text{-}meas}$) is obtained by replacing P_{aw} with P_{trach}.

$$R_{pt\text{-}meas} = \frac{P_{trach} - P_{plateau}}{\text{flow}}. \quad [2]$$

Without a direct measurement, P_{trach} is unknown but can be estimated by using published constants (Fig. 1) for R_{ett} (ie, $R_{ett\text{-}est}$). Estimated patient resistance ($R_{pt\text{-}est}$) is calculated

$$R_{pt\text{-}est} = R_{rs} - R_{ett\text{-}est}.$$

$$R_{pt\text{-}est} = \frac{P_{trach\text{-}est} - P_{plateau}}{\text{flow}}. \quad [3]$$

Rearranging:

$$P_{trach\text{-}est} = R_{pt\text{-}est} \times \text{flow} + P_{plateau} \quad [4]$$

Results are expressed as bias ± precision unless otherwise stated, according to the method of Bland and Altman.[8] For the catheter validation, agreement was evaluated by calculating bias (mean difference between lateral and catheter pressure measurements) and precision (standard deviation of the mean of the differences between the two measurements). For the patient investigations, bias ± precision were calculated to compare the simultaneously obtained values of estimated P_{trach} to measured P_{trach}, using the same methods. Bias was plotted against the mean of estimated P_{trach} and measured P_{trach}. We arbitrarily defined clinically acceptable limits of agreement as ± 3.0 cm H_2O. In Group-2 patients, analysis of variance was used to compare the bias and precision of P_{trach} at the 4 different flows. A value of $p < 0.05$ was considered statistically significant.

Results

Catheter Validation

Validation results of the catheter testing (lateral pressure minus test catheter pressure) produced a bias and precision of 1.71 ± 2.09 cm H_2O (Catheter 1), 1.14 ± 2.61 cm H_2O (Catheter 2), and 1.36 ± 2.24 cm H_2O (Catheter 3) when peak pressures up to 94 cm H_2O were developed in the test lung. In other words, the mean difference between lateral and test catheter pressure measurements was always ≤ 1.71 cm H_2O, with the test catheter reading lower than the sidearm reading.

Patient Data

We studied 20 patients with various diagnoses, admitted to our intensive care unit for management of acute respiratory failure. All patients were orally intubated with either an 8.0- or 8.5-mm ETT (with the exception of one 7.5-mm

tube) and had a mean intubation time of 5.9 days. ETTs were cut to lengths varying from 23 to 28 cm. The mean C_{rs} was 41.8 ± 10.9 mL/cm H_2O. A Puritan Bennett 7200ae ventilator was used for all patients except one who was ventilated with a Bear 1000.

Group 1

The mean values for measured P_{trach} and estimated P_{trach} in each patient are shown in Table 1. When P_{trach} was estimated from the ventilator P_{aw}, the mean bias and precision between measured and estimated P_{trach} was −9.9 ± 6.87 cm H_2O (range −22 to 2). However, when proximal P_{aw} was used, both bias and precision improved (−2.0 ± 3.5 cm H_2O). With predetermined acceptable limits of agreement at ± 3.0 cm H_2O, these results appear acceptable; however, the range was −8.0 to 3 cm H_2O, and the negative bias appeared to increase with increasing flows.

Table 1. Group 1—Estimated vs Measured P_{trach} Using Both Ventilator and Proximal Pressures at Different Flows

Flow	$P_{trach\text{-}est}$ (ventilator)	$P_{trach\text{-}est}$ (proximal)	P_{trach} (measured)	Bias (measured-ventilator)	Bias (measured-proximal)
60	27	26	29	2	3
80	34	30	26	−8	−4
80	34	28	27	−7	−1
85	27	23	23	−4	0
90	40	28	27	−13	−1
110	44	35	28	−16	−7
110	38	33	33	−5	0
120	52	47	39	−13	−8
120	50	27	28	−22	1
120	44	34	31	−13	−3
Mean Difference				−9.9 (6.87)	−2.0 (3.5)

Group 2

Group-2 patients were studied to examine the effect of inspiratory flow on estimated P_{trach}. The results for Group-2 patients are shown in Table 2. In the 10 Group-2 patients, the mean bias and precision ranged from −1.0 ± 1.58 to −10.2 ± 3.83 when peak P_{aw} was read from the ventilator. The mean bias was clearly dependent on the inspiratory flow, increasing as the flow increased ($p < 0.05$ for flows of 100 and 120 L/min when compared to 60 and 80 L/min). The mean bias and precision decreased, however, to −1.2 ± 2.17 to −3.6 ± 7.09 when proximal P_{aw} was used, and no dependence on flow was seen.

Discussion

In this study, we have demonstrated that clinically important and statistically significant differences exist between actual and estimated P_{trach} when ventilator pressures are used to estimate P_{trach}. Both bias and precision improve when measured proximal P_{aw} is used to estimate P_{trach}, but results are still not clinically acceptable at higher flows. Because the additional equipment needed to measure proximal P_{aw} is similar to that needed to directly measure P_{trach}, there is probably no advantage to measuring proximal P_{aw}.

Although Guttman et al[9] have reported a computer-assisted method of accurately calculating P_{trach}, it is complex; and we believe it to be impractical for routine clinical use. We are not aware of any other direct comparisons between estimated and measured P_{trach}. However, other authors[10,11] have compared in-vitro R_{ett} values with in-vivo measurements and found that in-vitro measurements of R_{ett} usually underestimate measurements taken in-vivo. Several explanations for differences between in-vitro and in-vivo measurements of R_{ett} have been given. In-vitro constants are usually determined on clean,

Table 2. Group 2—Mean Values (Standard Deviations) for Measured and Estimated P_{trach}, and the Mean Bias ± Precision for All Patients Studied. Data Using Pressures both from Ventilator and from True Proximal Site to Estimate P_{trach}.

	Flow (L/min)			
	60	80	100	120
P_{trach} cm H_2O (measured)	32.0 (6.60)	33.2 (3.11)	33.2 (2.78)	32.6 (2.70)
P_{trach} cm H_2O (estimated from ventilator P_{aw})	33.0 (5.83)	38.2 (3.03)	40.8 (4.44)*	42.8 (5.80)*
P_{trach} cm H_2O (estimated from proximal P_{aw})	33.2 (5.36)	35.2 (3.31)	36.8 (5.17)	36.2 (5.26)
bias ± precision (using ventilator P_{aw})	−1.0 ± 1.58	−5.0 ± 0.71	−7.6 ± 1.95	−10.2 ± 3.83
bias ± precision (using proximal P_{aw})	−1.2 ± 2.17	−2.0 ± 1.87	−3.6 ± 5.68	−3.6 ± 7.09

* $p < 0.05$ compared to flows of 60 and 80 L/min

straight ETTs at room temperature—in contrast to sputum-contaminated, curved ETTs at body temperature. Therefore, one would expect in-vivo values to be higher than those found in vitro, and higher values for R_{en} would be reflected in higher peak ventilator pressures. Warters et al[12] directly measured P_{trach} in the synchronized intermittent mandatory ventilation and the pressure control modes and found no difference in P_{trach} between modes in spite of a difference in P_{aw} measured by the ventilator. As with previously mentioned studies, they found that the ETT can also account for large discrepancies between P_{aw} and P_{trach}. Our contribution has been to show that P_{trach} cannot be accurately predicted from proximal pressures in many clinical situations.

Both in clinical practice and in recent studies,[13,14] practitioners and researchers continue to focus on proximal P_{aw} in the volume control mode of ventilation, ignoring P_{trach} and making $P_{plateau}$ a secondary measurement. We believe that this makes it difficult to interpret the data and draw conclusions.

Conclusions

In summary, we have shown that there can be large discrepancies between airway pressures taken from the ventilator, from a proximal site, and from a distal tracheal site, with the ventilator P_{aw} reading higher. Similarly, the ETT can account for large pressure drops in the system. If one is compelled to observe and follow peak P_{aw}, it should not be done from the ventilator but from a direct measurement of P_{trach}. However, it is even simpler to use $P_{plateau}$ as the sole parameter for preventing or minimizing barotrauma and ignore the peak P_{aw} from the ventilator. We agree with Warters et al[12] that "when airway pressures are monitored coincident with flow, intratracheal monitoring better reflects alveolar pressure than [does] proximal monitoring."

PRODUCT SOURCES

Test Lung:
Michigan Instruments, Grand Rapids MI

Cytology Brush:
Boston Scientific Corp, Boston MA

Jet Adapter:
Portex, Markham, Ontario, Canada

Pressure Transducers:
Validyne MP45-32-871, Northridge CA

Data-Acquisition System:
Direct, Ray Tech, Vancouver, BC

Parabolic Resistors, #3.9:
Michigan Instruments, Grand Rapids MI

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Jon Nilsestuen PhD RCP RRT, Robert Harwood MSA RRT, Ken Hargett BS RCP RRT, Section Editors. Submissions from readers are encouraged. Please follow the Manuscript Preparation Guide and submit to RESPIRATORY CARE Journal Office, 11030 Ables Lane, Dallas, Texas 75229.

A Case of Too Much Flow

Ken Hargett BS RRT, Rusty Reid RRT CPFT, and Jon Nilsestuen PhD RRT

After extubation in the operating room, a 63-year-old man with severe emphysema was admitted to the Surgical Intensive Care Unit (ICU) following lung volume reduction surgery via bilateral bovine stapling through median sternotomy. The patient tolerated the procedure well and was discharged to the General Care Unit. On postoperative Day 6, he complained of shortness of breath and required increasing levels of supplemental oxygen. A chest radiograph revealed a left pneumothorax. A left chest tube was inserted, and the patient was transferred back to the Surgical ICU. Because of continued shortness of breath and increased oxygen requirements, the patient was placed on a Nellcor Puritan Bennett 7200ae ventilator (Nellcor Puritan Bennett, Pleasanton CA). Initial ventilator settings were synchronized intermittent mandatory ventilation (SIMV) 10 breaths/min; F_{IO_2} 0.6; tidal volume (V_T) 0.650 L; and positive end-expiratory pressure (PEEP) 5 cm H_2O. After intubation, a chest radiograph revealed a persistent left pneumothorax and pnuemomediastinum. An additional chest tube was inserted mediastinally.

The patient stabilized on the ventilator with near normal blood gases. The patient's F_{IO_2} was decreased to 0.4 and the ventilator rate was decreased to 4 breaths/min. As is customary at our institution, the physicians ordered the addition of 10 cm H_2O of pressure support when the rate was decreased. Figure 1 reveals the pressure and volume waveforms from the 7200ae.

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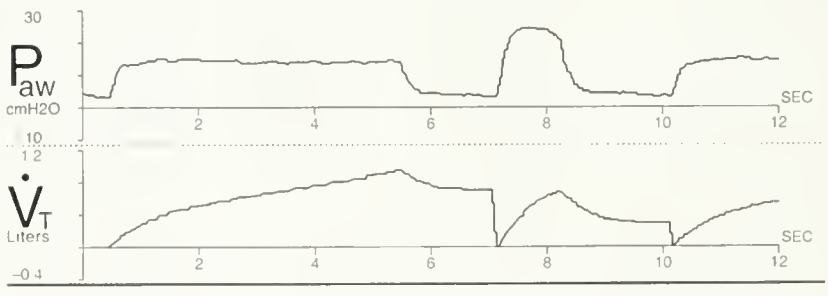

Fig. 1. The pressure and volume waveform taken from the Nellcor Puritan Bennett 7200ae graphic display. Ventilator settings include synchronized intermittent mandatory ventilation (SIMV) 4 breaths/min; pressure support 10 cm H_2O; tidal volume (V_T) 0.650 L; positive end-expiratory pressure (PEEP) 5 cm H_2O; and a decelerating flow pattern.

How would you answer these questions?

What has changed from Figure 1 regarding the time interval?

Can you identify the pressure support breaths and the SIMV breaths?

What is unusual about the flow waveform of the pressure support breaths?

Is anything unusual about the timing of the SIMV breaths?

Answers on next page

Answers

Types of Breaths. The first breath is a 10 cm H_2O pressure-support breath. The pressure plateau is at 15 cm H_2O pressure. The second breath represents the SIMV volume breath with a peak inspiratory pressure of 26 cm H_2O.

The First Breath. The inspiratory volume delivered by the pressure-support breath is greater than the 0.650 L set SIMV breath. The pressure-support breath shows an excessively long inspiratory time. Close inspection reveals a pressure plateau of 15 cm H_2O, that is held throughout the inspiratory time of 5 seconds. The volume waveform of the first breath also indicates that a substantial portion of the inspired volume was not exhaled. The actual volume of the 'leak' is indicated by the vertical line at the end of volume waveform of the first breath.

The Second Breath. This breath shows the much shorter inspiratory time of the SIMV mechanical breath. It also shows less volume on expiration than on inspiration. The 'leak' on the mandatory breath is much smaller than the leak during the pressure support breath.

The patient continued to complain of shortness of breath; therefore, the SIMV rate was increased to 10 breaths/minute. Because of the unusual waveform seen in Figure 1 the graphic display was reconfigured to show volume and flow (Fig. 2).

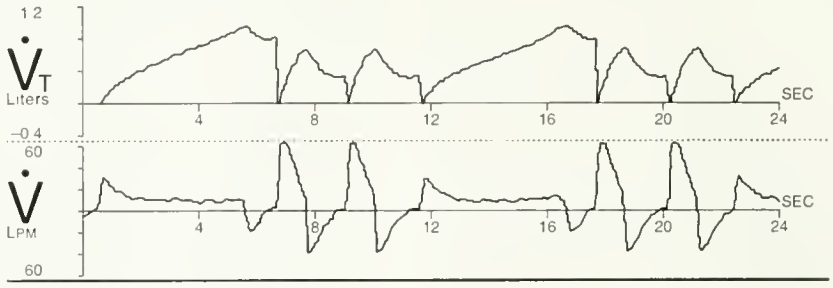

Fig. 2. The volume and flow waveform taken from the 7200ae using synchronized intermittent mandatory ventilation (SIMV) 10 breaths/min; pressure support 10 cm H_2O; tidal volume (V_T) 0.650 L; positive end-expiratory pressure (PEEP) 5 cm H_2O; and a decelerating flow pattern.

How would you answer these questions?

What has changed from Figure 1 regarding the time interval?

Can you identify the pressure support breaths and the SIMV breaths?

What is unusual about the flow waveform of the pressure support breaths?

Is anything unusual about the timing of the SIMV breaths?

Answers on page 1188

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Answers

Time Interval. The time interval in Figure 2 is now 24 seconds, allowing a greater number of breaths to be displayed.

Breaths. The first breath is a pressure-support breath followed by 2 SIMV breaths. This sequence is then repeated.

Flow Waveform. The flow waveform shows that flow decelerates to a constant value in the pressure support breaths. The inspiratory time is excessive and remains at 5 seconds.

SIMV Breaths. Even though the inspiratory volume is large in the pressure-support breaths the expiratory portion of the flow and volume waveform shows a much smaller exhalation as compared to the volume breaths in SIMV. Two SIMV breaths occur together suggesting autocycling.

Observation of the patient indicated that the chest tubes bubbled extensively during inspiration. At that time, the chest tubes were attached to suction. After discussion with the attending physician, the suction was removed and the chest tubes were left to water seal. An extensive leak was still evident. Changes in the PEEP level also had very little effect on the leak. Evaluation of the ventilator graphics led us to the conclusion that the pressure-support breaths were excessively long and that the air leak was larger on those breaths than on the volume breaths. Closer examination of the graphics indicated that the flow on the pressure-supported breaths never decelerated to the inspiratory flow termination criteria. Unlike most other ventilators, the primary cycling mechanism for the 7200ae on pressure-support breaths is when the flow decelerates to a value below 5 L/min. In this case the patient's air leak was greater than 5 L/min. The ventilator remained in the inspiratory phase until the secondary inspiratory cycling criteria was met. On the Nellcor Puritan Bennett 7200ae this criteria is 5 seconds.[1] The result was the prolonged inspiratory phase, which worsened the air leak and prevented the air leak from closing.[2] The majority of inspiratory volume was lost through the chest tubes. Studies have indicated that this lost volume does affect CO_2 removal and makes it difficult to determine the patient's contribution to effective minute ventilation.[3] It has been reported that patients with extensive air leaks can maintain abnormally low P_{CO_2} even while apneic.[4]

In an effort to reduce the inspiratory time and lost volume during pressure-support breaths, the pressure-support level was reduced from 10 to 8 cm H_2O. We did this because the peak inspiratory flow during pressure-supported breaths is determined by the target pressure and there might be a critical opening pressure for the air leak.[5] Figure 3 was generated after pressure support was reduced.

Fig. 3. Volume and flow waveforms taken from the 7200ae following the reduction from 10 cm H_2O to 8 in pressure support. Ventilator settings remained the same: synchronized intermittent mandatory ventilation (SIMV) 10 breaths/min; tidal volume (V_T) 0.650 L; positive end-expiratory pressure (PEEP) 5 cm H_2O; and a decelerating flow pattern.

How would you answer this question?

What change is seen in the second and third pressure-support breaths as compared to the first pressure-support breath?

Answer on page 1190

—1998—
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Answer

Breaths. The first breath continues to show prolonged inspiratory time and failure of the flow to reach the 5 L/min inspiratory termination criteria. The subsequent pressure-support breaths appear to reach less than 5 L/min and cycle into expiration normally.

The pattern seen in Figure 3 continued sporadically. Some pressure-support breaths cycled normally, and others continued to be prolonged breaths with extensive leaks. We believed that the pressure support was deleterious to the patient and offered no benefit in regards to comfort or minute ventilation. Therefore, the use of pressure support was discontinued, and we assessed the waveform (Fig. 4).

Fig. 4. Volume and flow waveforms taken from the 7200ae with no pressure support. The ventilator settings were synchronized intermittent mandatory ventilation (SIMV) 10 breaths/min; tidal volume (V_T) 0.650 L; positive end-expiratory pressure (PEEP) 5 cm H_2O; and F_{IO_2} 0.4.

How would you answer these questions?

Can you identify the spontaneous breaths in the flow waveform?

What is unusual about the flow waveforms on these spontaneous breaths?

What is the explanation for the two consecutive SIMV breaths?

Answers on page 1192

1997 Literary Awards

From The American Respiratory Care Foundation

Allen DeVilbiss Technology Paper Award

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A Comparison of the i-STAT System and Corning 278 for the Measurement of Arterial Bood Gas Values
[Respir Care 1997;42(7):693–697]

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Paul B Blanch BA RRT
& Michael Banner PhD RRT

Is Tracheal Pressure Ventilator Control Comparable to an Equivalent Level of Pressure Support Ventilation?
[Respir Care 1997;42(11):1022-1033]

Radiometer Awards for Best Feature Articles

Katherine A Douglas BEd RRT

Blood Gas Corner — *Premature Infant Born by Emergency Cesarean Section*
[Respir Care 1996;41(12):1126-1129]

Samer Alkhuja MD

Test Your Radiologic Skill — *Bilateral Upper Lobe Cavities: The Lesions Are Obvious but What about the Cause?*
[Respir Care 1997;42(11):1045-1047]

Janice Benedetti CRNA
& Charles G Durbin Jr MD

Graphics Corner — *Abnormal Capnogram during Laparoscopic Cholecystectomy in a Lung-Transplant Patient*
[Respir Care 1997;42(2):227-228]

Answers

Spontaneous Breaths. The graphic shows 4 SIMV breaths that are characterized by the rise in pressure coinciding with the decelerating flow pattern. The graphic also demonstrates several spontaneous breaths that are characterized by inspiratory flow with only a slight decrease in baseline pressure. These breaths are unusual in that expiratory flow is not present and, probably, secondary to the chest tube leak.

SIMV Breaths. The 2 adjacent SIMV breaths are due to autocycling. The leak from the chest tube results in a slight pressure drop that triggered the ventilator.

Sensitivity was adjusted first to 2 then 3 cm H_2O below baseline; however, the ventilator continued the same pattern. We decided to place the ventilator in flow-by. Initially with flow-by set to 10 L/min and the sensitivity set at 3 L/min, the ventilator continued to autocycle. Base flow was then increased to 20 L/min and the flow sensitivity was incrementally increased to 4 then 5, then 6, and, finally, 7 L/min. The flow sensitivity setting had to be greater than the air leak in order to prevent the ventilator from autocycling (Fig. 5).

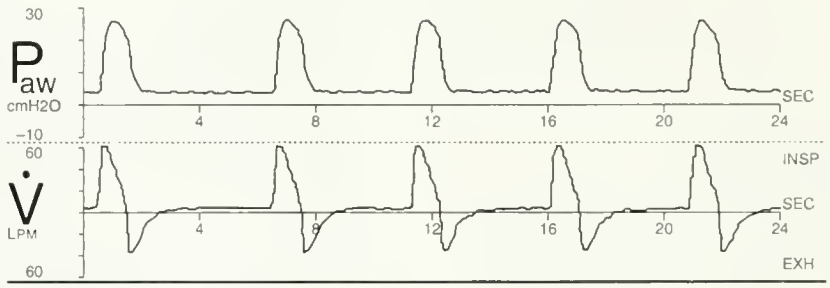

Fig. 5. The waveforms obtained with the addition of flow-by and the ventilator settings: synchronized intermittent mandatory ventilation (SIMV) 10 breaths/min; tidal volume (V_T) 0.650 L; positive end-expiratory pressure (PEEP) 5 cm H_2O; and F_{IO_2} 0.4.

How would you answer these questions?

How has the SIMV pattern changed as compared to Figure 4?

What is unusual about the flow pattern between the SIMV breaths?

Answers & Disctussion on page 1194

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Answers

SIMV Patterns. The SIMV breaths are now evenly spaced with the absence of autocycling. The flow waveform shows the presence of a continuous flow between each of the mandatory breaths.

Flow Pattern. Close inspection of the pressure waveform shows very small changes in airway pressure that result from the patient's spontaneous efforts. This was verified by observation of the patients respiratory efforts. The patient is breathing from the 20 L/min flow-by in the circuit so these efforts do not simultaneously appear as changes in the flow waveform.

Discussion

The patient's leak gradually reduced and the patient was eventually weaned from the ventilator. Because of the flow termination criteria of 5 L/min, the use of pressure support in patients with extensive chest tube leaks can cause the leak to persist. As a result of this case a new ventilator management protocol is being developed at our hospital for these patients. The protocol will incorporate graphic analysis to define proper ventilator function. Investigations are under way to determine the effect of PEEP and pressure-support levels on the presence and amount of leak in lung volume reduction surgery.

This example shows the importance of graphic analysis in proper application of ventilator settings and use of modes. In this particular case the use of pressure support was not indicated due to the large air leak and the specific pressure-support termination criteria of 5 L/min. Most other ventilators use an algorithm of 25% of initial peak inspiratory flow. This would have resulted in proper operation of pressure support in this case. Newer generation ventilators have user-adjustable termination criteria for pressure-supported breaths. It will be important for the clinician to be able to intelligently use graphic capabilities of the ventilator to properly select the inspiratory termination criteria that is best for the patient's clinical condition.

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RETROSPECTROSCOPE REDUX:

Man-Cans (Conclusion)

The Body Plethysmograph (Body Box)

Since the mid 1950s, the term body plethysmograph (or body box) has taken on a new meaning. There are many models and varieties, but essentially the box is a chamber with an airtight door and an internal volume of about 600 liters, designed so that a normal subject or patient can sit or stand comfortably in it (or, in some models, lie down). A large number of measurements can be made on the patient in a few minutes, without inserting any needles or catheters; or as we now say in our best (or worst) jargon, "The interventions are non-invasive"! These measurements include thoracic gas volume, airway resistance, pulmonary tissue resistance, trapped gas volume, volume of gas in the gastrointestinal tract, and instantaneous pulmonary capillary blood flow (19-24).

Like earlier methods of the 1880s (15, 16), the present-day body box also depends on Boyle's law (17), although the new method does not use Pflüger's technic of rapid decompression. Figure 4 shows schematically how Boyle's law is now used to measure thoracic gas volume, and Figure 5 shows how it is used to measure airway resistance.

Because I was responsible for the initial development of the present-day box, it's only fair for you to ask me why I was addle-brained or stubborn enough (or both) to work on a body box in 1948 when Pflüger and other German scientists had already tried and abandoned the idea, and when the most respected pulmonary clinical physiologist of the 1930s and 1940s (Ronald Christie) said of it, "The apparatus is cumbersome and its management full of technical difficulties, while more cooperation is demanded of the subject than can be given by even the most healthy individuals" (18).

Because I've written an essay entitled "Tell It Like It Was," I'll tell it that way. The answer is that I was neither addle-brained nor stubborn. I couldn't have been because I had not *read* (or remembered) the page in Christie's article dealing with the "pneumatometric method," although I had read and re-read the other 14 pages dealing with his new gas-dilution technic. And in 1948 I had not read the article by Pflüger and some later ones also written in German.

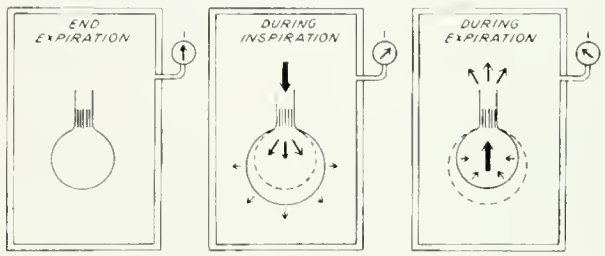

Fig. 4. The rectangle is an airtight body box. The patient (represented by his alveoli and conducting airway) breathes air about him. The two circles with pointers represent pressure gauges; one measures pressure in the box, and the other measures airway pressure, which equals alveolar pressure when there is no air flow. A: at end-expiration alveolar pressure equals atmospheric pressure; V is unknown. B: the airway is then occluded, and the patient inspires; this creates a new gas volume ($v + \Delta V$) and a new pressure, P'. Knowing P, P', and ΔV, V (initial, unknown thoracic gas volume) can be calculated using Boyle's law, $PV = P'V'$.

Why hadn't I? I was six years old when the United States entered World War I against Germany, and I lived in York, Pennsylvania, a city that despite its English name, had a large "Pennsylvania Dutch" (really *Deutsch*) population. I recall that most of our neighbors spoke in "Dutch" over their white-washed back-yard fences and anti-"Dutch" feelings ran so high in 1917-18, at least among youngsters, that not even dachshunds or German shepherd dogs were safe on the streets. So, when I went to high school, French was the popular foreign language, and I took three years of French and no German. When I went to college in the early 1930s, anti-German feelings from World War I had died out, but again I studied French, largely because I had no intention of going into science and felt no need for learning German. Then came the rise in Germany of Hitler, Nazism, anti-intellectualism, and book burning, and scientists outside of Germany came to have a low regard for then current German science. As a result, the popularity of German as a language declined again. Later, I did learn a little German in summer school, but not enough to make reading scientific German easy or simple. If German had been my sec-

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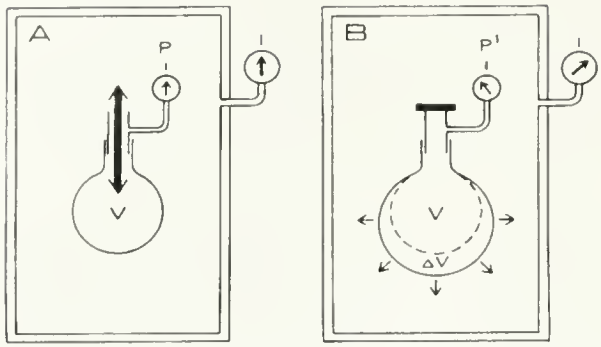

Fig. 5. The patient (only the lung is pictured) is seated within an airtight box; pressure is measured continuously in the box around the patient. One would expect that inspiration of 500 mL of air from the box into the patient's lungs would produce no pressure fluctuations in the box (if precautions are taken to prevent changes due to changes in temperature and humidity of the respired gas); actually, the box pressure rises during inspiration. This is simply because, at the beginning of inspiration, muscular action has enlarged the thorax and has lowered alveolar pressure below atmospheric pressure. Throughout inspiration, alveolar gas (previously at atmospheric pressure) is now at subatmospheric pressure and so occupies more volume; this is the same as adding this increment of gas volume (resulting from the decompression) to the plethysmograph, and so the pressure rises. The pressure change is registered by a very sensitive manometer. The reverse happens during expiration, when alveolar gas is compressed. From this measured pressure and appropriate calibrations, alveolar pressure can be calculated for any moment in the respiratory cycle. From these simultaneous measurements of alveolar pressure and flow (pnuemotachygraph), airway resistance can be determined.

ond language, I would never have worked on the body box, for I would have known that everyone who had, gave it up!

Ancel Keys and Body Volume

Where, then, did I get the idea of using a body plethysmograph? From studies on fat people by Ancel Keys and Josef Brožek, who never used a body box to study the lungs! Keys and Brožek were interested in the study of body leanness and fatness (25, 26), an interest that eventually led Keys to examine fat in the diet and the relationship between cholesterol and atherosclerosis. But in the late 1940s, Keys and his group were measuring body fatness by using Archimedes' principle—that the apparent loss of weight of an object in water is exactly equal to the weight of fluid displaced by the object. So Keys weighed his subjects first in air and then submerged in water; the difference between the two results was the volume of the man. Weight/volume equals specific gravity, and tables of specific gravity that allow a correction for bone (which has a specific gravity of 2.0 and makes up 4 per cent of total body mass) permitted a calculation of body fat, which has a specific gravity well below 1.0.

But total immersion in water is not a quick or pleasant way of determining body volume, so Keys turned from Archimedes to Boyle. He constructed an elegant stainless steel twin chamber in the shape of a split-level sarcophagus. The upper part, 300 liters in volume, was large enough that Keys could slide in a patient and still close the door; the lower part, 15 liters in volume, could be connected to the larger one in an instant. When a subject was in the larger compartment, both compartments were filled with air, but at different pressures; when connected, the two pressures quickly equalized and from this final pressure (and of course Boyle's law), Keys could calculate the volume of the man in the 300-liter compartment.

As far as I can tell, the authors never published more than one line on this method, and this in an abstract (27): "Body volume may also be estimated by its effect on the pressure relations in a closed air system of known size." They obviously ran into technical difficulties that they could not overcome and abandoned their man-can.

However, they first presented a demonstration of their beautifully constructed apparatus and the proposed method for measuring *body* volume at the 1948 meetings of the American Physiological Society in Minneapolis. I was there and saw it. I wasn't interested in body fatness (although with advancing age, I now am) or in measuring body volume, but I *was* interested in rapid, quickly repeatable measurements of lung function. And I realized that the Keys apparatus might well be the key to measuring lung volume (or more accurately, thoracic gas volume) and instantaneous and continuous alveolar pressure (a necessary datum for calculating airway resistance in humans).*

Our First Man-Can

On my return from Minneapolis, Dr. Stella Botelho and I put together some inexpensive, makeshift parts to produce a man-can. The top was a heavy lucite cone (it came from a discarded fighter plane); it sat on a flanged metal vat and once the subject climbed inside, we could quickly seal the cone to the vat. We worked out the theory (see figures 4 and 5) and knew that it must work, but we ran into many technical problems. These included trying to measure volumes and pressures of air while the temperature and water vapor pressure of the air were changing both continuously and cyclically; drift in our electrical capacitance manometers† (when

*I'm not sure whether Keys's group knew that their method might be used to measure residual lung volume in humans. It was essential to their studies of body fatness because the "Archimedes method" included air in the lungs, and they could at that time correct for the volume of air (which has an extremely low specific gravity) only be referring to tables (28). They did recognize a potential error in the existence of gastrointestinal gas because they noted "the air pressure system also allows the estimation of gas in the digestive tract..."(27).

†The strain gauge was first reported in 1947 and used then for measuring systemic arterial blood pressure. In the late 1950s, it became commercially available as a rugged, stable, and dependable transducer to measure either low or high pressures.

they were functioning, which was about half the time): creaking floors; inadequate recording apparatus; and a less than ideally designed man-can.

Stella Botelho realized that the problem with changing temperature and water vapor pressure could be licked if the subject was surrounded by air at body temperature and saturated with water vapor. And she observed that the error in measurement increased when the subject breathed more deeply and decreased when breathing was shallow. I'm sure she suggested that the error might be tolerable if the subject panted, with rapid shallow breathing, and I'm equally sure I squelched the idea on the grounds that panting was not a physiologic type of breathing for man. We were getting close, but not quite close enough.

In any case, by 1950 we knew that if we were going to continue, the lucite cone and metal vat* must go, and we must design a better box.

We went to the well-known ———————— Instrument Company whose management expressed total uneoncern with building a prototype for us.† So in late 1950, we turned to our medical school shop for a new model.

It took more than a year for its completion. During that time, Stella Botelho returned to her real love, the study of nerve-muscle function, and later to the mechanism and regulation of lacrimal secretion (in which fields she has become a leader), and I worked on the reflex effects of 5-OH-tryptamine (serotonin), then a brand-new and very exciting compound. Some of our fellows worked a bit with the new box, but when their tenure ended, someone put a sign, "Comroe's Folly," on it and closed the door. There were many suggestions for using it—as a bombproof shelter, a summer storage place to protect winter coats from moths, and a pay telephone booth (to recoup our investment).

Role of the National Heart Institute

In the meantime, several important events had occurred. The first was that, on June 16, 1948, President Truman signed the National Heart Act and created the National Heart Institute (which later became the National Heart and Lung Institute and is now the National Heart, Lung, and Blood Institute). The 1948-49 budget for the new Institute included an unusual item—several million dollars for construction of new research facilities to be awarded without any requirement for matching funds. The National Advisory Heart Council, an all-star cast headed by Paul Dudley White and including Irvine Page, Tinsley Harrison, T. Duckett Jones, Conrad Elvehjem, and Mary Lasker, held its first meeting on September 8, 1948 and its second, three months later, on December 10-11. Between the first and second Council meetings, the Institute asked for and received applications from various institutions for construction grants for research laboratories. Council members visited some or all of these sites and rated them so that they could make awards at the December meeting.

This first event was a well planned one. The second event, my learning of the existence of these funds, was completely fortuitous. The Xerox machine had not been invented by 1948, and information regarding National Institutes of Health programs did not automatically reach every faculty member whether he wanted it or not, as it does now. However, the dean of the School of Medicine at the University of Pennsylvania knew about the funds and he informed the chairman of the school's research committee, so at least two people in Philadelphia knew about them. Although I disliked serving on committees, I *was* on the research committee that passed on the suitability of extramural grant applications before they left the University. At one of these meetings in the fall of 1948, at the end of a long, dreary session, the Chairman announced something to this effect: "It's too late now to apply because the deadline to mail applications is only a few days away, but you'll be interested to know that the new National Heart Institute has allocated money for constructing research facilities." I pricked up my ears and said, "Why too late? We can write a pretty convincing application that will document our needs and do it within a day." The Chairman answered, "Ah, indeed you could, but each application for *construction* funds must be accompanied by full applications for *research grants* as well." That seemed to be a crunching blow, but I got together Seymour Kety, Bob Dripps, and Ward Fowler, and in less time than it takes me to write this sentence, we decided to apply for construction funds and buttress our application with four research grant applications. We made the deadline and that December we were awarded about $160,000 for new laboratories and funds for all four of our research projects.‡ The $160,000 bought for us half the eighth floor of a new pavilion in the University Hospital (completed in 1952) and a home for the new body box, also delivered in 1952.

DuBois and Box Number 2

The third important event happened in 1952 and only because the National Heart Institute had provided additional space for our research. We offered an assistant professorship to Arthur DuBois, then a resident in medicine at Peter Bent Brigham Hospital in Boston; he accepted and joined our staff in July of 1952. Arthur had worked in Wallace Fenn's lab-

*I'm told that the vat served a very useful function for at least the next decade; the workmen in the vivarium used it for dipping and sterilizing animal cages!

†Today, at least five scientific instrument companies manufacture body plethysmographs including one with a space-age design in a heavenly powder-blue color.

‡You'll be interested to know that the average amount of our research grants was $15,000 per year!

oratory at the University of Rochester as a research fellow in 1949-1951, measuring alveolar carbon dioxide concentrations with Richard Fowler's rapid infrared analyzer, but for the first year or so with our group at Penn he studied mechanical properties of the lungs and thorax, using forced oscillations applied to the chest wall.

Arthur had (and still has) a highly original, creative, and innovative mind. When we were together at Penn, he always brought a completely fresh approach to any problem he tackled. (He later received a National Institutes of Health Research *Career* Award and is now director of the Pierce Foundation Laboratory at Yale.) Like most creative individuals, he preferred to work on his own problems; I'm pretty sure that he would not have worked happily on an assigned problem. Arthur was also (and still is) an unusually gentle person and would not intrude on another's work.

Arthur still says that I offered him a position only because he was a good electrician, and that the only space available for his work in 1952 was the janitor's broom closet. But in 1953-1954, space was ready for him in our new hospital research laboratories (those built with the National Heart Institute's funds), and he got the lab that was storing "Comroe's Folly." In late 1953, with the box staring him in the face all day long, with no one with a claim to it, and no one *telling* him to work on it, Arthur went into action. A few weeks later, he walked into my office and, with an obviously suppressed smile of satisfaction, said softly, "I've got the body box working and I've made some measurements of airway resistance in man."

Our records show that Arthur made his first measurements of airway resistance on Ferdinand Kreuzer, a fellow physiologist from Holland, on January 16, 1954, and that by February 15, in my report to the Army Chemical Corps, I was able to include data on airway resistance in 7 normal subjects and 25 patients. He reported his work to the American Society for Clinical Investigation that spring (29). I believe that Arthur "got the box working" in a few weeks, but delayed for a while telling me of his success to spare me the embarrassment of having worked on it a few years, off and on, without being able to solve the technical difficulties.

What did Arthur do that we had not? He solved the water vapor-temperature problem by having the subject *pant* at low tidal volumes that kept the air moving back and forth within the heated flow meter, at body temperature, and saturated with water vapor. He realized that, because resistance = pressure difference/air flow, it is a *ratio* and can be measured over and over again as a *slope* of a cathode-ray oscilloscope used as an X-Y plotter; panting produced rapidly repeated slopes that, on a persistent screen, gave a mean value. Rapid measurements, plus repeated venting of the box (to lick the problem of drift) and frequent calibration of the chamber (with the patient inside) with an oscillating pump, licked other problems. He seemed to have a gift for making the Lilly manometer work and he made calculations easy by circling the face of the cathode ray screen with direct-reading scales (30).

I've been praised many times since for using unmatched Machiavellian cunning in picking that particular spot to store the body box, especially since Arthur was already working on pulmonary resistance and the response of the lungs-thorax system to oscillating pressure waves. If I "tell it like it was," I'm not cunning. If I deserve praise, it is for *not* giving Arthur the assignment to "make the box work;" I knew he liked to pick his own problems and in good time, he picked this one.

A few months later, Grant Lee, a young cardiologist and clinical physiologist from Oxford, arrived on a 6-month fellowship. We had an absolute rule of *never* accepting a fellow for less than a year and preferably for not less than two years. This was our exception to the rule and a fortunate exception, for Lee was loaded with enthusiasm, energy, and ability. Lee wrote to me recently:

> I arrived in Philadelphia on the weekend of the Spring meetings at Atlantic City in April 1954. Only Arthur was in the lab; all the rest had gone to Atlantic City. He asked me why I had come to the department and I said that I was interested in the pulmonary capillary circulation as I had found in man no evidence to support the idea that there was significant reflex vasoconstriction in the lungs.
>
> Arthur then promptly asked the following question: "If you consider there to be little vaso-motor activity in the pulmonary arterial system, you must imply that there is little peripheral resistance. Do you think lung capillary blood flow is pulsatile or smooth? If it is pulsatile it could prejudice current assumptions about gas diffusion in the lungs (Bohr equilibration)." Without thinking I said that of course I thought capillary blood flow must be pulsatile. He promptly replied, "then let's measure it."

Arthur DuBois had studied Krogh and Lindhard's 1912 method of measuring pulmonary blood flow by determining uptake of nitrous oxide (Seymour Kety had made all of us nitrous-oxide conscious—and occasionally unconscious) and had already made some preliminary calculations of the pressure drop that would occur in our box when gaseous nitrous oxide in alveoli became dissolved nitrous oxide in blood flowing through the pulmonary capillaries. DuBois and Lee went to work; they finished their experiments by July 6, 1954 and presented the data to the American Physiological Society two months later (31). In rapid succession, DuBois and colleagues then completed studies on thoracic gas volume (19), volume of trapped gas in the lung (22), pulmonary tissue resistance (21), and volume of gas in the gastrointestinal tract (23).

Gastrointestinal Gas

The first workers to use a man-can to measure the volume of gastrointestinal gas were Blair, Bates, and Fenn in 1943 (32), although the first full report was by Blair, Dern, and Bates in 1947 (33). They too made use of Boyle's law; when their

subjects voluntarily compressed their abdominal gas by a forced expiratory effort, the team measured the change in intragastric pressure concomitant with a change in box pressure. Their paper will be long remembered because it reveals one component of a closely guarded secret: the volume of CO_2 released at 37° C from 133 mL of Coca-Cola® at atmospheric pressure is 490 mL! Their subjects drank 133 mL of this mysterious concoction as an easy and guaranteed way of adding 490 mL of gas to the stomach during the experiments; this is worth remembering by patients who suffer from abdominal distention.*

Blair had found that healthy subjects have about 1,000 mL of gas in their stomach and intestines. DuBois and his colleagues, using their box in 1955, found values of only 100 mL of gas. They believed that the larger values found by Blair were due to artifacts caused by making measurements at the end of a forced expiratory effort instead of at resting lung volume, as in the 1955 study.

Now that many hospitals have pulmonary function laboratories and each of these usually houses a body plethysmograph, it would be interesting to know how many times gastroenterologists, working under the same roof, have requested that their pulmonary colleagues measure the volume of gastrointestinal gas in patients.

Low-Pressure Chambers

In the 1800s, physicians became interested in therapeutic measures that involved either placing patients in special chambers, in which total atmospheric pressure could be decreased or increased, or inhalation by their patients of a wide variety of gases, vapors, or mists. These probably were stimulated by the opening, in Bristol, England, in 1798, of Thomas Beddoes's "Pneumatic Institution." This, considered by Barzun (36) to be the first clinical research laboratory, had as its goal the study of inhalation treatment of respiratory diseases. The latter part of the 18th century had seen the discovery of oxygen, hydrogen, nitrogen, and illuminating gas. Beddoes himself, aided by James Watt, designed what seems to have been the first O_2 tent and his superintendent, Humphry Davy, had used hydrogen to measure his own lung volume and also suggested the use of nitrous oxide as an analgesic-anesthetic. Beddoes's clinical trials must also have brought tribulations because he once remarked, "Those who decry experiments in medicine do not perhaps perceive that they [those who decry] cut off all hope from those at present incurable."

Beddoes cannot be held responsible for those who later carried aerotherapeutics too far. One of these was Junod, in 1835. The 1909 edition of Albutt and Rolleston's System of Medicine has a chapter on artificial aerotherapeutics and this contains a subsection on "artificial atmospheres varying in barometric pressure" in which C. T. Williams (see reference 12, Part One, AMERICAN REVIEW OF RESPIRATORY DISEASE, November 1977) discusses Junod's use of a rarefied atmosphere as a therapeutic agent. Junod devised a hollow copper sphere, about 1.4 meters in diameter, in which he could place a man and decrease the atmospheric pressure by one third (from 760 mm Hg to about 500 mm Hg). When his patients, instead of getting better, became short of breath and turned blue, Junod gave up low pressure applied to the whole of a man and concentrated on a low-pressure boot applied to a leg (which engorged the limb's blood vessels, but did not increase blood *flow* through them) and on cupping glasses that applied suction to a localized area of the skin. As late as 1909, Albutt and Rolleston wrote about cupping:

> From the ease and readiness of its application it is of value in cases in which blood is to be drawn rapidly from an organ, and especially in haemoptysis. I have witnessed some of the most profuse haemorrhages from the lung suddenly brought to a termination by the use of dry cupping to the chest wall; and I have often observed that so long as the cupping-glasses are kept on, the haemorrhage has been controlled, and that on their removal it may return. Hence the great advantage of cupping-glasses with exhausters attached by which a partial vacuum can be maintained. This treatment, if carefully applied, leaves no mark, and it is to be preferred to blistering or strong poulticing in cases of gouty disposition or where the patient has a very irritable skin.

A negative pressure chamber was also used widely by early thoracic surgeons to prevent collapse of a lung when they opened a patient's thorax. Sauerbruch's 1904 chamber was the first in a long series of these (37). The patient's head (outside the chamber) was at atmospheric pressure (760 mm Hg); his trunk (inside the chamber and the object of surgical attack) was kept at 750 mm Hg. The 10 mm Hg difference in pressure kept the lung continuously inflated, although it did nothing to provide for rhythmic ventilation. The cumbersome negative pressure chamber died when surgeons (or anesthesiologists) rediscovered the efficacy of intermittent positive-pressure ventilation applied to the nose and mouth or endotracheal tube.

High-Pressure Chambers

The scientific study of humans exposed to high atmospheric pressures began with Paul Bert, who, between 1870 and 1885, studied humans at low and high pressures and recorded his

*It should be noted that DuBois, when a research fellow in Fenn's laboratory in 1949-51, frequently passed Blair's body box (stored in the hallway!) without its arousing in him any intense desire to use it. And that Arthur Otis, another of the Fenn team, published an article on measuring alveolar pressure in humans in 1948 (34), but used an interrupter instead of Blair's body box to do it. And Fenn himself, studying both cats and newborn babies in a body box in 1955, noted rhythmic variations in box pressure with inspiration and expiration, but used them to measure tidal volume and not alveolar pressure (35).

work in a massive volume, *La Pression Barométrique* (38). The English translation runs to 1178 pages, of which 522 give a detailed history of previous work, 518 present Bert's own experiments, and 118 are his résumé and conclusions! Bert had worked under Claude Bernard and in 1869 succeeded him as professor of physiology at the Faculté des Sciences in Paris. Among Bert's many contributions were settling the cause of mountain sickness (he proved that it was due to low partial pressure of oxygen and not to a decrease in *total* barometric pressure), demonstrating the key role of oxygen pressure or tension in determining the amount of oxygen combined with hemoglobin, obtaining data for the first O_2 and hemoglobin association (or dissociation) curve, and learning the effects of high atmospheric pressures on humans (caisson disease) and the causes of decompression sickness.

The first suggestion that inhalation of oxygen at more than 1 atmosphere of pressure might be useful came from the great respiratory physiologist, J. S. Haldane (39). The earlier work of Henry and of Dalton had shown that the amount of gas that dissolves in a liquid depends upon the partial pressure of the gas to which the liquid is exposed, and Bert had shown that even when hemoglobin becomes saturated with O_2, the plasma in blood can accept still more O_2 in physical solution. Haldane reasoned that carbon monoxide might not be toxic to cells in general, but might kill animals only by preventing oxygen from combining with hemoglobin (carbon monoxide has an affinity for hemoglobin more than 200 times that of O_2 for hemoglobin). He knew that when the partial pressure of O_2 was 100 mm Hg, 0.3 mL of O_2 dissolved in 100 mL of the watery parts of blood (whether hemoglobin was present or not); if he increased the partial pressure of O_2 to 2,300 mm Hg (about 3 atmospheres), 6.9 mL of O_2 should dissolve in plasma, enough to meet the needs of body tissues without their drawing off any O_2 from the great store of oxygen chemically combined with hemoglobin. So he gave mice a lethal concentration of carbon monoxide to breathe, which, once in the bloodstream, formed carbon monoxide hemoglobin and replaced oxyhemoglobin. Haldane then saved the lives of the mice by exposing them to oxygen in a chamber at a pressure of 3 atmospheres.

Despite this clear-cut demonstration, Oxygen at High Pressures (OHP) was not used clinically, even in treating carbon-monoxide poisoning, for many years. Chambers were huge, clumsy, and expensive and never in the right place when needed. And other physiologists demonstrated that breathing O_2 at sea level could be toxic to lungs, eyes, and brain, and that exposure to OHP could cause severe symptoms, even death.

However, pressure chambers hung on. L. H. Gray (40) showed in 1953 that the sensitivity of cancer cells to x-rays was decreased by anemia, and increased by oxygen, particularly by OHP. So some radiotherapy departments began to treat cancer patients with combined radiotherapy and OHP. Then, when open-heart surgery came along (but the artificial heart-lung was not yet ready for routine use), surgeons needed ways of prolonging the survival of a patient when his heart had been deliberately stopped to permit the surgeon to repair it. Hypothermia (to decrease the patient's need for blood, by cooling the entire body) and OHP (to provide a store of dissolved O_2 in plasma and tissues) came into use. I believe that Boerema's eye-catching phrase, "life without blood" (41), aroused considerable interest in OHP, and soon chambers were in use to treat infections with anaerobic bacteria, to save patients with carbon-monoxide poisoning, to limit the extent of myocardial infarcts or to decrease the irritability of ischemic cardiac tissue, to decrease brain damage resulting from obstruction to cerebral arteries, and to treat blue babies. And medicine now had two new types of specialists: the "intensivist," whose domain was the intensive care unit, and the "hyperbaricist," who operated the OHP chamber.

But there is still much to learn besides the toxic effects of OHP and how to avoid them by the posting of safe time limits for each level of OHP. Of great importance, of course, is the knowledge that O_2 is not a substitute for blood, except for short periods. Flowing blood carries not only O_2 to tissues but also the substrates of energy (glucose, fatty acids) and regulators of cell function; flowing blood also has a function in removing excess CO_2, H^+, lactate, and other materials from cells. O_2 is neither energy nor a source of energy; O_2 *is* essential but only as the terminal electron acceptor in a complex mitochondrial electron transport system that generates adenosine triphosphate. Few cells have any stores of O_2. Many cells have some intracellular stores of substrate that can keep things going if O_2 is available, but once these stores are depleted, the only source of substrate is the structure of the cell itself; eating into this, of course, leads to cellular deterioration and death, even though cells are saturated with oxygen.

Concluding Remarks

"Man-Cans" is a story of ideas, failures, and successes. It would be nice to have nothing but success and no failures in science; some persons, hoping to achieve this, lay down policies for the advancement of scientific knowledge and set up programs guaranteed to do so. However, Harry Eagle once remarked that he had more confidence in ten thousand individual, working scientists than in a committee of ten especially wise ones. This is a bit of an exaggeration, of course, but it makes his point that biomedical knowledge comes in unexpected and unpredictable ways, and often, or usually, from any one of the ten thousand "undirected" scientists, including some not generally regarded as biomedical scientists. The story of Man-Cans bears this out:

- Boyle's law was around for several centuries during which it had many practical applications, but not to medicine. It was a chance stroll by an "undirected" individual through a scientific exhibit in 1948 that led to the development of a body box for measuring airway resistance and thoracic

gas volume. Ten very wise men would not have thought of this because Brožek and Keys, who exhibited their sarcophagus there, never put their ideas into print. Why didn't they? Because the ideas didn't work out as they had hoped.

- Though there are no hard data to support my beliefs, I suspect that most important discoveries—ones that have had real impact on the forward direction of research—have come from one of the ten thousand scientists who had their own ideas and wanted to work on them, instead of from those "directed by ten wise men" to work on projects. The collapse of German science during the 1930s and 1940s, though highly respected previously, is a classic example. The wise men turned out not to be so wise, and there were no ideas from individuals. Totally directed German science between 1930-1945 even had the effect of diminishing the impact and utilization of splendid German science published long before the 1930s.

- "Man-Cans" deals in large part with apparatus and devices whose construction requires a knowledge of engineering, or at least mechanical and electronic know-how. Yet these were developed largely by individuals working in the machine shops of universities of medical schools (e.g., the iron lung, developed by a Harvard engineer, Phillip Drinker, working with mechanics in the medical school machine shop). They were rarely the result of accurate predictions by wise captains of industry; indeed, some captains turned down proffered opportunity to develop prototypes.

- True, the ideas of many of the ten thousand individuals do fail, but even in failing, and sometimes *because* of failing, they advance science. It often takes a mistake by one man (or now even by one woman) to interest another person in setting things straight. Possibly my concepts are wrong; if so, I hope they will goad someone to get the hard facts to disprove them.

Julius H Comroe, Jr.

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Chest Trauma Simulation

Simulates a 62-year-old male involved in a motor vehicle accident. The user must perform initial assessment and recommend initial therapy to stabilize the patient. In the course of the treatment, the patient undergoes surgery for a thoracotomy and is then placed on a mechanical ventilator. The user is asked to recommend initial ventilator settings. Subsequent decision-making sections include recommending fiberoptic bronchoscopy, making further ventilator changes based on ABG values, and evaluating the patient for possible extubation.

Item SP10 $65

Status Asthmaticus Simulation

Involves the initial assessment of a 35-year-old male with a history of allergic asthma. Low flow oxygen is administered initially, followed by bronchodilator therapy. As the simulation progresses, the patient is intubated and receives mechanical ventilation. The user is required to make initial settings and adjustments according to ABG results and patient response. Also included in the simulation is a switch to IMV mode, sedation, and eventual extubation and placement on a 40 percent aerosol mask.

Item SP12 $65

Drug Overdose Simulation

This simulation involves an 18-year-old male entering the ER intoxicated, with a possible drug overdose. The user performs initial assessment that includes ventilatory parameters. During the course of the simulation, the patient is transferred to the ICU for further monitoring, then deteriorates and requires oxygen therapy via a 40 percent T-piece and mechanical ventilation. The patient is gradually weaned and extubated with supplemental oxygen therapy.

Item SP13 $65

Chronic Obstructive Pulmonary Disease (COPD) Simulation

The user is asked to perform initial assessment and pulmonary function studies on a patient with chronic lung disease. Then, the user classifies the patient's disease as mild, moderate, severe restrictive, and/or obstructive disease. The simulation asks for home care and home equipment cleaning recommendations. Three months later the patient enters the ER with respiratory distress, requiring initial evaluation and therapy.

Item SP14 $65

Acute Respiratory Distress Syndrome (ARDS) Simulation

This simulation presents a 30-year-old male 36 hours after emergency surgery for excision of a large duodenal ulcer; the patient is now restless and complaining of dyspnea. The simulation proceeds through initial oxygen therapy, followed by increasing F_{IO_2}, and then CPAP. The patient is eventually intubated and placed on a mechanical ventilator, where deteriorating ABGs and static compliance call for increasing PEEP, which is gradually increased to 25 cmwp. ABGs, compliance, and shunt studies are used to evaluate the effectiveness of the PEEP therapy.

Item SP15 $65

Epiglottitis Simulation

Presents a 4-year-old female entering the ER with a "cold" and elevated temperature. A lateral neck x-ray reveals massive edema of the epiglottis, whereupon the child is taken to surgery and intubated. The user is asked to recommend appropriate airway and size for the patient. After further assessment, CPAP is initiated with room air, and two days later the patient is evaluated for extubation and postextubation therapy.

Item SP16 $65

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Premature Neonate Simulation

Includes initial assessment of a 15-year-old, high-risk, pregnant female upon admission and initial assessment of the baby after birth. APGAR scores of 4 and 3 are obtained in the assessment, and the simulation proceeds through adjustment of CPAP and F_{IO_2} for the infant. The infant does not receive mechanical ventilation and is eventually assessed for extubation, extubated, and given supplemental oxygen.

Item SP17 $65

Head Trauma Simulation

Simulates the initial evaluation and recommendations for care of a 25-year-old involved in a motor vehicle accident. After evaluation in the ER, the patient is taken to the OR for a craniotomy. Assessment following surgery (including measurement of ICP) indicates the need for CMV, whereupon the user is required to evaluate the effect of CMV, hyperinflation, and ET suctioning on ICP and take appropriate steps to maintain a normal ICP.

Item SP18 $65

Muscular Dystrophy Simulation

This is a simulation of a 10-year-old with diagnosed muscular dystrophy who is entering the ER with respiratory distress. The simulation asks for patient assessment and initial therapy recommendations. Diagnostic tests are then presented to evaluate the degree of muscle involvement, and the user is required to recommend further therapy based on the results of these tests. As the simulation progresses, IPPB is required and the user is asked to provide instructions to the patient on IPPB treatment. CPT is also involved, with the correct positioning of the patient required.

Item SP19 $65

Chest Trauma II Simulation

Involves a motor-vehicle-accident patient with multiple trauma. Initial assessment followed by oxygen therapy occurs in the ER, followed by further assessment leading to intubation and placement on a mechanical ventilator. Based on blood gas results, several ventilator changes are made. The patient suffers a pneumothorax while on the ventilator, and the user is required to assess and recommend treatment for the condition. In addition, the simulation includes measurement and clinical application of compliance.

Item SP110 $65

Neonate II Simulation

Begins with the assessment of a neonate delivered by C-section who is receiving oxygen in an incubator. Based on the assessment, the infant in intubated and placed on CPAP, but problems occur, requiring extubation and bag ventilation. After reintubation, the CPAP level is changed based on ABG results and the user is required to evaluate the infant for extubation. In the simulation, the patient then fails the procedure and the user must evaluate the patient to determine the cause of the problem, which is diagnosed as a diaphragmatic hernia.

Item SP111 $65

Cystic Fibrosis Simulation

This simulation presents a 13-year-old patient with cystic fibrosis in the outpatient clinic. The initial task is to perform a pulmonary assessment, after which a decision must be made concerning action to be taken based on the assessment. Following obtainment of additional diagnostic information, the patient is admitted, but the user is asked to set up a regimen of home care therapy, including recommendations about chest physiotherapy, oxygen therapy, aerosol therapy, and disinfecting home care equipment. The child is admitted two weeks later for further assessment and recommendations regarding treatment.

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Pulmonary Rehabilitation Simulation

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Item PE6

Sources of Error in the Determination of Blood Gas Values and pH
Item PE7

Temperature Adjustment of Blood Gases and pH
Item PE8

Measurement of Static Compliances and Dynamic Characteristics Curves During Mechanical Ventilation
Item PE9

Arterial Blood Gas Interpretation
Item PE10

An Introduction to Hemodynamic Monitoring
Item PE11

Physical Assessment
Item PE12

Smoking Cessation

Nicotine Dependency Evaluation and Treatment
Item SC1

Bedside Counseling of the Hospitalized Smoker
Item SC2

To order call the AARC at (972) 243-2272

MEDWATCH

THE FDA MEDICAL PRODUCTS REPORTING PROGRAM

For VOLUNTARY reporting by health professionals of adverse events and product problems

Page ___ of ___

Form Approved OMB No 0910-0291 Expires: 4/30/98
See OMB statement on reverse

FDA Use Only (Resp Care)

Triage unit sequence #

A. Patient information

1 Patient identifier	2. Age at time of event: or Date of birth:	3. Sex	4 Weight
In confidence		☐ female ☐ male	___ lbs or ___ kgs

B. Adverse event or product problem

1 ☐ Adverse event and/or ☐ Product problem (e.g., defects/malfunctions)

2. Outcomes attributed to adverse event (check all that apply)

☐ death (mo day yr)
☐ life-threatening
☐ hospitalization – initial or prolonged
☐ disability
☐ congenital anomaly
☐ required intervention to prevent permanent impairment/damage
☐ other

3. Date of event (mo day yr)	4 Date of this report (mo day yr)

5. Describe event or problem

6 Relevant tests/laboratory data, including dates

7. Other relevant history, including preexisting medical conditions (e.g., allergies, race, pregnancy, smoking and alcohol use, hepatic/renal dysfunction, etc.)

C. Suspect medication(s)

1 Name (give labeled strength & mfr/labeler, if known)
#1
#2

2 Dose, frequency & route used
#1
#2

3 Therapy dates (if unknown, give duration) from/to (or best estimate)
#1
#2

4 Diagnosis for use (indication)
#1
#2

5 Event abated after use stopped or dose reduced
#1 ☐ yes ☐ no ☐ doesn't apply
#2 ☐ yes ☐ no ☐ doesn't apply

6 Lot # (if known)
#1
#2

7 Exp. date (if known)
#1
#2

8. Event reappeared after reintroduction
#1 ☐ yes ☐ no ☐ doesn't apply
#2 ☐ yes ☐ no ☐ doesn't apply

9 NDC # (for product problems only)

10 Concomitant medical products and therapy dates (exclude treatment of event)

D. Suspect medical device

1 Brand name

2 Type of device

3 Manufacturer name & address

4 Operator of device
☐ health professional
☐ lay user/patient
☐ other.

5. Expiration date (mo/day/yr)

6
model #
catalog #
serial #
lot #
other #

7 If implanted, give date (mo/day/yr)

8 If explanted, give date (mo/day/yr)

9 Device available for evaluation? (Do not send to FDA)
☐ yes ☐ no ☐ returned to manufacturer on ___ (mo/day/yr)

10 Concomitant medical products and therapy dates (exclude treatment of event)

E. Reporter (see confidentiality section on back)

1 Name & address

phone #

2 Health professional?
☐ yes ☐ no

3. Occupation

4 Also reported to
☐ manufacturer
☐ user facility
☐ distributor

5 If you do NOT want your identity disclosed to the manufacturer, place an " X " in this box. ☐

FDA

Mail to: MEDWATCH
5600 Fishers Lane
Rockville, MD 20852-9787

or FAX to:
1-800-FDA-0178

FDA Form 3500 1/96)

Submission of a report does not constitute an admission that medical personnel or the product caused or contributed to the event.

ADVICE ABOUT VOLUNTARY REPORTING

Report experiences with:

- medications (drugs or biologics)
- medical devices (including in-vitro diagnostics)
- special nutritional products (dietary supplements, medical foods, infant formulas)
- other products regulated by FDA

Report SERIOUS adverse events. An event is serious when the patient outcome is:

- death
- life-threatening (real risk of dying)
- hospitalization (initial or prolonged)
- disability (significant, persistent or permanent)
- congenital anomaly
- required intervention to prevent permanent impairment or damage

Report even if:

- you're not certain the product caused the event
- you don't have all the details

Report product problems – quality, performance or safety concerns such as:

- suspected contamination
- questionable stability
- defective components
- poor packaging or labeling
- therapeutic failures

How to report:

- just fill in the sections that apply to your report
- use section C for all products except medical devices
- attach additional blank pages if needed
- use a separate form for each patient
- report either to FDA or the manufacturer (or both)

Important numbers:

- 1-800-FDA-0178 to FAX report
- 1-800-FDA-7737 to report by modem
- 1-800-FDA-1088 to report by phone or for more information
- 1-800-822-7967 for a VAERS form for vaccines

If your report involves a serious adverse event with a device and it occurred in a facility outside a doctor's office, that facility may be legally required to report to FDA and/or the manufacturer. Please notify the person in that facility who would handle such reporting.

Confidentiality: The patient's identity is held in strict confidence by FDA and protected to the fullest extent of the law. The reporter's identity, including the identity of a self-reporter, may be shared with the manufacturer unless requested otherwise. However, FDA will not disclose the reporter's identity in response to a request from the public, pursuant to the Freedom of Information Act.

The public reporting burden for this collection of information has been estimated to average 30 minutes per response, including the time for reviewing instructions, searching existing data sources, gathering and maintaining the data needed, and completing and reviewing the collection of information. Send comments regarding this burden estimate or any other aspect of this collection of information, including suggestions for reducing this burden to:

DHHS Reports Clearance Office
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Hubert H. Humphrey Building, Room 531-H
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Washington, DC 20201

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U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service • Food and Drug Administration

FDA Form 3500-back

Please Use Address Provided Below – Just Fold In Thirds, Tape and Mail

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Food and Drug Administration
Rockville, MD 20857

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MEDWATCH
The FDA Medical Products Reporting Program
Food and Drug Administration
5600 Fishers Lane
Rockville, MD 20852-9787

American Association for Respiratory Care

Membership Application

Please read the eligibility requirements for each of the classifications in the right-hand column, then complete the applicable section. All information requested below must be provided, except where indicated as optional. See other side for more information and fee schedule. Please sign and date application on reverse side and type or print clearly. Processing of application takes approximately 15 days.

- ☐ Active
- Associate
  - ☐ Foreign
  - ☐ Physician
  - ☐ Industrial
- ☐ Special
- ☐ Student

Last Name _____

First Name _____

Social Security No. _____

Home Address _____

City _____

State _____ Zip __ __ __ __ __ - __ __ __ __

Phone No. (_____) _____

Primary Job Responsibility (check one only)

- ☐ Technical Director
- ☐ Assistant Technical Director
- ☐ Pulmonary Function Specialist
- ☐ Instructor/Educator
- ☐ Supervisor
- ☐ Staff Therapist
- ☐ Staff Technician
- ☐ Rehabilitation/Home Care
- ☐ Medical Director
- ☐ Sales
- ☐ Student
- ☐ Other, specify _____

Type of Business

- ☐ Hospital
- ☐ Skilled Nursing Facility
- ☐ DME/HME
- ☐ Home Health Agency
- ☐ Educational Institution
- ☐ Manufacturer or supplier
- ☐ Other, specify _____

Date of Birth (optional) _____ Sex (optional) _____

U.S. Citizen? _____ Yes _____ No

Have you ever been a member of the AARC? _____

If so, when? From _____ to _____

Preferred mailing address: ☐ Home ☐ Business

For office use only

FOR ACTIVE MEMBER

An individual is eligible if he/she lives in the U.S. or its territories or was an Active Member prior to moving outside its borders or territories, and meets ONE of the following criteria: (1) is legally credentialed as a respiratory care professional if employed in a state that mandates such; OR (2) is a graduate of an accredited educational program in respiratory care; OR (3) holds a credential issued by the NBRC. An individual who is an AARC Active Member in good standing on December 8, 1994, will continue as such provided his/her membership remains in good standing.

PLEASE USE THE ADDRESS OF THE LOCATION WHERE YOU PERFORM YOUR JOB, NOT THE CORPORATE HEADQUARTERS IF IT IS LOCATED ELSEWHERE

Place of Employment _____

Address _____

City _____

State _____ Zip __ __ __ __ __ - __ __ __ __

Phone No. (_____) _____

Medical Director/Medical Sponsor _____

FOR ASSOCIATE OR SPECIAL MEMBER

Individuals who hold a position related to respiratory care but do not meet the requirements of Active Member shall be Associate Members. They have all the rights and benefits of the Association except to hold office, vote, or serve as chair of a standing committee. The following subclasses of Associate Membership are available: Foreign, Physician, and Industrial (individuals whose primary occupation is directly or indirectly devoted to the manufacture, sale, or distribution of respiratory care equipment or supplies). Special Members are those not working in a respiratory care-related field.

PLEASE USE THE ADDRESS OF THE LOCATION WHERE YOU PERFORM YOUR JOB, NOT THE CORPORATE HEADQUARTERS IF IT IS LOCATED ELSEWHERE

Place of Employment _____

Address _____

City _____

State _____ Zip __ __ __ __ __ - __ __ __ __

Phone No. (_____) _____

FOR STUDENT MEMBER

Individuals will be classified as Student Members if they meet all the requirements for Associate Membership and are enrolled in an educational program in respiratory care accredited by, or in the process of seeking accreditation from, an AARC-recognized agency.

SPECIAL NOTICE — Student Members do not receive Continuing Respiratory Care Education (CRCE) transcripts. Upon completion of your respiratory care education, continuing education credits may be pursued upon your reclassification to Active or Associate Member.

School/RC Program _____

Address _____

City _____

State _____ Zip __ __ __ __ __ - __ __ __ __

Phone No. (_____) _____

Length of program

- ☐ 1 year
- ☐ 2 years
- ☐ 4 years
- ☐ Other, specify _____

Expected Date of Graduation (REQUIRED INFORMATION)

Month _____ Year _____

AARC American Association for Respiratory Care • 11030 Ables Lane • Dallas, TX 75229-4593 • (972) 243-2272 • Fax (972) 48 2720

Demographic Questions

We request thot you onswer these questions in order to help us design services ond progroms to meet your needs.

Check the Highest Degree Earned

- ☐ High School
- ☐ RC Groduote Technicion
- ☐ Associote Degree
- ☐ Bochelor's Degree
- ☐ Moster's Degree
- ☐ Doctorote Degree

Number of Years in Respiratory Care

- ☐ 0-2 yeors
- ☐ 3-5 yeors
- ☐ 6-10 yeors
- ☐ 11-15 Yeors
- ☐ 16 yeors or more

Job Status

- ☐ Full Time
- ☐ Port Time

Credentials

- ☐ RRT
- ☐ CRTT
- ☐ Physicion
- ☐ CRNA
- ☐ RN
- ☐ LVN/LPN
- ☐ CPFT
- ☐ RPFT
- ☐ Perinotol/Pediotric

Salary

- ☐ Less thon $10,000
- ☐ $10,001-$20,000
- ☐ $20,001-$30,000
- ☐ $30,001-$40,000
- ☐ $40,000 or more

PLEASE SIGN

I hereby opply for membership in the Americon Associotion for Respirotory Core ond hove enclosed my dues. If opproved for membership in the AARC, I will obide by its bylows ond professionol code of ethics. I outhorize investigotion of oll stotements contoined herein ond understond thot misrepresentotions or omissions of focts colled for is couse for rejection or expulsion.

A yeorly subscription to RESPIRATORY CARE journol ond *AARC Times* mogozine includes on ollocotion of $6.50 from my dues for eoch of these publicotions.

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Signature _____

Date _____

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Poyment must occompony your opplicotion to the AARC. Fees ore for 12 months. (NOTE: Renewol fees ore $65.00 Active, Associote-Industriol or Associote-Physicion, or Speciol stotus; $80.00 for Associote-Foreign stotus; ond $35.00 for Student stotus).

☐ Active	$77.50
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☐ Associote (Foreign)	$92.50
☐ Speciol	$77.50
☐ Student	$35.00
TOTAL	$_____

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Estoblished to recognize the speciolty oreos of respirotory core, these sections publish o newsletter four times o yeor thot focuses on issues of specific concern to thot speciolty. The sections olso design the speciolty progromming ot the notionol AARC meetings.

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☐ Diognostics Section	$5.00
☐ Continuing Core-Rehobilitotion Section	$5.00
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TOTAL	$_____

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☐ Totol Amount Enclosed $_____

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- ☐ Viso

Cord Number

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Cord Expires _____/_____

Signoture _____

Mail application and appropriate fees to:

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RESPIRATORY CARE

Manuscript Preparation Guide

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Publication Categories & Structure

Research Article: A report of an original investigation (a study). It includes a Title Page, Abstract, Introduction, Methods, Results, Discussion, Conclusions, Product Sources, Acknowledgments, References, Tables, Appendices, Figures, and Figure Captions.

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Review Article: A comprehensive, critical review of the literature and state-of-the-art summary of a pertinent topic that has been the subject of at least 40 published research articles. Title Page, Outline, Introduction, Review of the Literature, Summary, Acknowledgments, References, Tables, Appendices, and Figures and Captions may be included.

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Letter: A signed communication, marked "For publication," about prior publications in this Journal or about other pertinent topics. Tables and illustrations may be included.

Blood Gas Corner: A brief, instructive case report involving blood gas values—with Questions, Answers, and Discussion.

Drug Capsule: A mini-review paper about a drug or class of drugs that includes discussions of pharmacology, pharmacokinetics, and pharmacotherapy.

Graphics Corner: A brief case report incorporating waveforms for monitoring or diagnosis—with Questions, Answers, and Discussion.

Kittredge's Corner: A brief description of the operation of respiratory care equipment—with information from manufacturers and editorial comments and suggestions.

PFT Corner: Like Blood Gas Corner, but involving pulmonary function tests.

Cardiorespiratory Interactions. A case report demonstrating the interaction between the cardiovascular and respiratory systems. It should be a patient-care scenario; however, the case—the central theme—is the systems interaction. CRI is characterized by figures, equations, and a glossary. See the March 1996 Issue of RESPIRATORY CARE for more detail.

Test Your Radiologic Skill: Like Blood Gas Corner, but involving pulmonary medicine radiography and including one or more radiographs; may involve imaging techniques other than conventional chest radiography.

Review of Book, Film, Tape, or Software: A balanced, critical review of a recent release.

Preparing the Manuscript

Print on one side of white bond paper, 8.5 in. x 11 in. (216 x 279 mm) with margins of at least 1 in. (25 mm) on all sides of the page. Use ***double-spacing*** throughout the ***entire*** manuscript. Use a standard font (eg, Times, Helvetica, or Courier) at least 10 points in size, and

do not use italics except for special emphasis. Number all pages in upper-right corners. Indent paragraphs 5 spaces. Do ***not justify***. **Do *not* put authors' names, institutional affiliations or allusions to institutional affiliations in the text, or other identification anywhere except on the title page.** Repeat title only (no authors) on the abstract page. Begin each of the following on a new page: Title Page, Abstract, Text, Product Sources List, Acknowledgments, References, each Table, and each Appendix. Use standard English in the first person and active voice.

Center main section headings on the page and type them in capital and small letters (eg, Introduction, Methods, Results, Discussion). Begin subheadings at the left margin and type them in capital and small letters (eg, Patients, Equipment, Statistical Analysis).

References. Cite only published works as references. Manuscripts accepted but not yet published may be cited as references: designate the accepting journal, followed by (in press), and provide 3 copies of the in-press article for reviewer inspection. Cite references in the text with superscript numerals. Assign numbers in the order that references are first cited. On the reference page, list the cited works in numerical order. Follow the Journal's style for references. Abbreviate journal names as in *Index Medicus*. List all authors.

Article in a journal carrying pagination throughout volume:

> Rau JL, Harwood RJ. Comparison of nebulizer delivery methods through a neonatal endotracheal tube: a bench study. Respir Care 1992;37(11):1233-1240.

Article in a publication that numbers each issue beginning with Page 1:

> Bunch D. Establishing a national database for home care. AARC Times 1991;15(Mar):61,62,64.

Corporate author journal article:

> American Association for Respiratory Care. Criteria for establishing units for chronic ventilator-dependent patients in hospitals. Respir Care 1988;33(11):1044-1046.

Article in journal supplement: (Journals differ in their methods of numbering and identifying supplements. Supply sufficient information to promote retrieval.)

> Reynolds HY. Idiopathic interstitial pulmonary fibrosis. Chest 1986; 89(3 Suppl):139S-143S.

Abstract in journal: (Abstracts citations are to be avoided. Those more than 3 years old should not be cited.)

> Stevens DP. Scavenging ribavirin from an oxygen hood to reduce environmental exposure (abstract). Respir Care 1990;35(11): 1087-1088.

Editorial in journal:

> Enright P. Can we relax during spirometry? (editorial). Am Rev Respir Dis 1993;148(2):274.

Editorial with no author given:

> Negative-pressure ventilation for chronic obstructive pulmonary disease (editorial). Lancet 1992;340(8833):1440-1441.

Letter in journal:

> Aelony Y. Ethnic norms for pulmonary function tests (letter). Chest 1991;99(4):1051.

Paper accepted but not yet published:

> Hess D. New therapies for asthma. Respir Care (year, in press).

Personal author book: (For any book, specific pages should be cited whenever possible.)

> DeRemee RA. Clinical profiles of diffuse interstitial pulmonary disease. New York: Futura; 1990. p. 76-85.

Corporate author book:

> American Medical Association Department of Drugs. AMA drug evaluations. 3rd ed. Littleton CO: Publishing Sciences Group; 1977.

Chapter in book with editor(s):

> Pierce AK. Acute respiratory failure. In: Guenter CA, Welch MH, editors. Pulmonary medicine. Philadelphia: JB Lippincott; 1977:26-42.

Tables. Use consecutively numbered tables to display information. Start each table on a separate page. Number and title the table and give each column a brief heading. Place explanations in footnotes, including all nonstandard abbreviations and symbols. Key the footnotes with conventional designations (*, †, ‡, §, ‖, ¶, **, ††) in consistent order, placing them superscript in the table body. Do not use horizontal or vertical rules or borders. Do not submit tables as photographs, reduced in size, or on oversize paper. Use the same typeface as in the text.

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Mail three copies [1 copy with author(s) name(s), affiliation(s), 2 copies without name(s) and affiliation(s) for reviewers] of the manuscript, figures, and 1 diskette, and the Cover Letter & Checklist to RESPIRATORY CARE, 600 Ninth Avenue, Suite 702, Seattle WA 98104. Do not fax manuscripts. Protect figures with cardboard. Keep a copy of the manuscript and figures. Receipt of your manuscript will be acknowledged.

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kreilkamp@aarc.org

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If yes, please describe. _____

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Yes No If yes, please disclose: _____

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- Has the accuracy of the references been checked, and are they correctly formatted?
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1998 Call for Abstracts

RESPIRATORY CARE • OPEN FORUM

The American Association for Respiratory Care and its science journal, RESPIRATORY CARE, invite submission of brief abstracts related to any aspect of cardiorespiratory care. The abstracts will be reviewed, and selected authors will be invited to present posters at the OPEN FORUM during the AARC International Respiratory Congress in Atlanta, Georgia, November 7–10, 1998. Accepted abstracts will be published in the October 1998 issue of RESPIRATORY CARE. Membership in the AARC is not required for participation. All accepted abstracts are automatically considered for ARCF research grants.

SPECIFICATIONS—READ CAREFULLY!

An abstract may report **(1) an original study, (2) the evaluation of a method, device or protocol, or (3) a case or case series.** Topics may be aspects of adult acute care, continuing care/rehabilitation, perinatology/pediatrics, cardiopulmonary technology, or health care delivery. The abstract may have been presented previously at a local or regional—but not national—meeting and should not have been published previously in a national journal. The abstract is the only evidence by which the reviewers can decide whether the author should be invited to present a poster at the OPEN FORUM. Therefore, *the abstract must provide all important data, findings, and conclusions.* Give specific information. Do not write such general statements as "Results will be presented" or "Significance will be discussed."

Essential Content Elements

Original study. Abstract *must* include (1) Background: statement of research problem, question, or hypothesis; (2) Method: description of research design and conduct in sufficient detail to permit judgment of validity; (3) Results: statement of research findings with quantitative data and statistical analysis; (4) Conclusions: interpretation of the meaning of the results.

Method, device, or protocol evaluation. Abstract *must* include (1) Background: identification of the method, device, or protocol and its intended function; (2) Method: description of the evaluation in sufficient detail to permit judgment of its objectivity and validity; (3) Results: findings of the evaluation; (4) Experience: summary of the author's practical experience or a lack of experience; (5) Conclusions: interpretation of the evaluation and experience. Cost comparisons should be included where possible and appropriate.

Case report. Abstract *must* report a case that is uncommon or of exceptional educational value and must include (1) Introduction: Relevant basic information important to understanding the case. (2) Case Summary: Patient data and response, details of interventions. (3) Discussion: Content should reflect results of literature review. The author(s) should have been actively involved in the case and a case-managing physician must be a co-author or must approve the report.

Abstract Format and Typing Instructions

Accepted abstracts will be photographed and reduced by 40%; therefore, the size of the original text should be *at least* 10 points. A font like Helvetica or Geneva makes the clearest reproduction. The first line of the abstract should be the title in all capital letters. Title should explain content. Follow title with names of all authors (including credentials), institution(s), and location; underline presenter's name. Type or electronically print the abstract *single spaced in a single paragraph in the space provided on the abstract blank.* Insert only one letter space between sentences. Text submission on diskette is encouraged but must be accompanied by a hard copy. Identifiers will be masked (blinded) for review. Data may be submitted in table form, *and simple figures may be included provided they fit within the space allotted.* No figures, illustrations, or tables are to be attached to the abstract form. Provide all author information requested. A clear photocopy of the abstract form may be used. Standard abbreviations may be employed without explanation; new or infrequently used abbreviations should be spelled out on first use. Any recurring phrase or expression may be abbreviated, if it is first explained. Check the abstract for (1) errors in spelling, grammar, facts, and figures; (2) clarity of language; and (3) conformance to these specifications. An abstract not prepared as requested may not be reviewed. Questions about abstract preparation may be telephoned to the editorial staff of RESPIRATORY CARE at (972) 406-4667.

Deadline Allowing Revision

Authors may choose to submit abstracts early. Abstracts postmarked by February 16, 1998 will be reviewed and the authors notified *by letter only* to be mailed by March 20, 1998. Rejected abstracts will be accompanied by a written critique that should, in many cases, enable authors to revise their abstracts and resubmit them by the final deadline (May 4, 1998).

Final Deadline

The mandatory Final Deadline is May 4, 1998 (postmark). Authors will be notified of acceptance or rejection *by letter only*. These letters will be mailed by July 20, 1998.

Mailing Instructions

Mail (Do not fax!) 2 clear copies of the completed abstract form, diskette (if possible), and a stamped, self-addressed postcard (for notice of receipt) to:

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1998 Respiratory Care Open Forum

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2. Follow title with all authors' names including credentials (underline presenter's name), institution, and location.
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4. **Do not use type size less than 10 points.**
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Calendar of Events

AARC & AFFILIATES

February 4–6, 1998—Oregon Society

Annual Pacific Northwest Respiratory Care Conference at the Valley River Inn in Eugene, Oregon. Events include a manager's breakfast featuring AARC Executive Director Sam Giordano.

Contact: Irene Iwata-Morgan at (503) 494-6158.

February 5–8, 1998—Clinical Laboratory Management Association (CLMA)

CLMA will co-host The Second Annual Congress on Integrated Health Care Management at the Dallas Lakes Hilton, Dallas, Texas. Last year's Congress was highlighted in the March issue of *AARC Times*.

Contact: CLMA at (610) 995-9580 or (202) 543-7971.

OTHER MEETINGS

February 8–11, 1998—American College of Chest Physicians

Cardiopulmonary Wellness and Rehabilitation meeting in Breckenridge, Colorado.

Contact: Arlene Karavich at (847) 498-1400 or fax (847) 498-5460.

March 1, 1998—Maryland Thoracic Society

The Maryland Thoracic Society, medical arm of the American Lung Association of Maryland, hosts its 38th Annual Meeting and Scientific Session at the Renaissance Harborplace Hotel in Baltimore, Maryland.

Contact: Anne Eder at (410) 560-2120 for fee information.

March 1–5, 1998—Children's National Medical Center

The 14th annual Children's National Medical Center symposium on "ECMO and Advanced Therapies for Respiratory Failure" will be held in Keystone, Colorado. Registration deadline is January 8, 1998.

Contact: Cynthia Poindexter at (202) 884-5018.

March 3–4, 1998—International Conference on Paediatric Asthma

Castle House Conferences is organizing the International Conference on Paediatric Asthma, A Masterclass Symposium, to be held at the Maastricht Exhibition and Congress Centre, Maastricht, The Netherlands.

Contact: Louise Sutherland in Kent, United Kingdom at +44 (0) 1892 539606, fax +44 (0) 1892 517773, E-mail inquiries @castlehouse.co.uk or http:// www.castlehouse.co.uk.

March 12–14, 1998—California Society for Pulmonary Rehabilitation & the California Society for Cardiac Rehabilitation

"Vital Connection: Heart & Lungs … a Partnership for Life" at the Long Beach Hilton, Long Beach, California.

Contact: Jim Barnett at (714) 365-2106 or Julia Kuwada at (310) 674-7050 ext 3316.

April 15–17, 1998—The University of Massachusetts Medical Center, the Pediatric Pulmonary & Cystic Fibrosis Center, & the National Association of Pediatric Home & Community Care

7th annual conference on "Needs" at the Regal Knickerbocker, Chicago, Illinois.

Contact: Contact Dorothy Page at (508) 856-1908 or fax (508) 856-2609, for fee information.

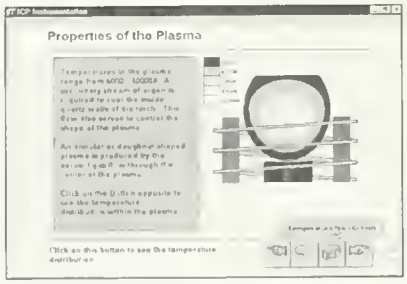
May 8–10, 1998—Medical University of South Carolina ECMO Service

8th annual Southeastern ECMO Conference at the Frances Marion Hotel, Charleston, South Carolina.

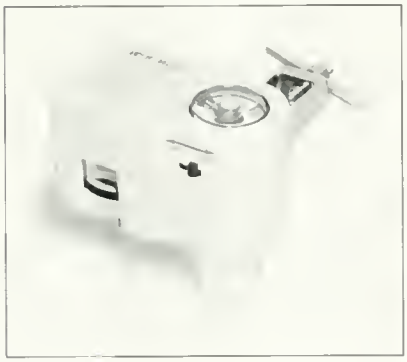
Contact: Sandra Ballard at (803) 792-9941.

New Products & Services

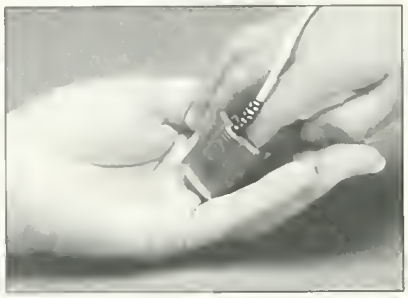
News releases about new products and services will be considered for publication in this section. There is no charge for these listings. Send descriptive release and glossy black and white photographs to RESPIRATORY CARE Journal, New Products and Services Dept, 11030 Ables Lane, Dallas TX 75229-4593. The Reader Service Card immediately follows the authors & advertisers index at the back of the Journal.



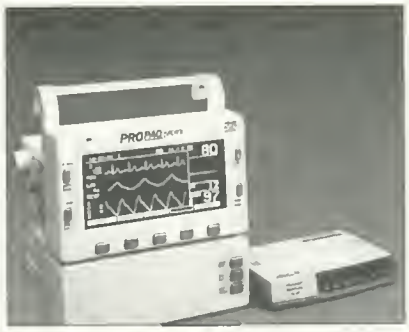
COMPUTER-BASED TRAINING. Fundamentals of Inductively Coupled Plasma Spectrometry is added to SAVANT's line of computer-based training software. The Windows®-based program offers a comprehensive course for entry-level or experienced workers, according to SAVANT. For a demonstration disk of the program or a SAVANT catalog, circle Reader Service Number 160.



OXYGEN CONSERVER. Victor Medical announces the introduction of a pneumatically driven, no-battery, single-tube cannula oxygen conserver; the O_2n Demand™ conserver is a complete unit, regulator, and conserver in one. According to the manufacturer, the patent-pending device uses no batteries and is maintenance-free. The conserver includes 5 flow settings, 1-3 L/min, in either continuous or conserve mode. The unit's flow dial is recessed to prevent the accidental change of flow. The conserver can also accept cylinder pressure up to 3,000 psi and is compatible with all CGA 540 valved and CGA 870 post-style cylinders. The O_2n Demand's optional shoulder system features a Universal bag, M-6 cylinder, cannula, and wrench. Circle Reader Service Number 161 for further information.

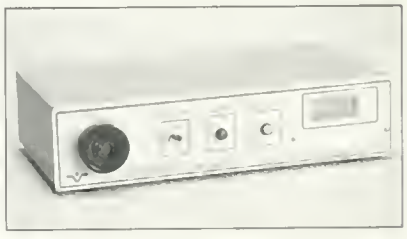


PEDIATRIC SENSOR. Nonin Medical Inc introduces the Pediatric Finger Clip Sensor, developed for use with Nonin pulse oximeters. Specifically created for the pediatric patient, the self-aligning finger clip is designed to minimize the effect of motion artifact while maximizing comfort and ease of use, the manufacturer claims. Circle Reader Service Number 162.



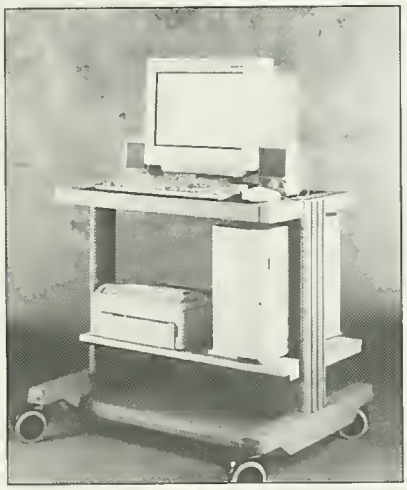
VITAL SIGNS MONITOR. Protocol Systems Inc introduces telephone modem communications capability for its Propaq® vital signs monitors. The Modem Propaq monitor is capable of transmitting patient data via a simple telephone modem to Protocol's Acuity® System central surveillance work station, giving health care providers the flexibility to remotely monitor patients located both inside and outside the hospital, according to the manufacturer. Circle Reader Service Number 163.

BRONCHODILATOR COMBINATION. Boehringer Ingelheim Pharmaceuticals Inc introduces Combivent® Inhalation Aerosol, a combination of bronchodilators (ipratropium bromide and albuterol sulfate) in a single metered dose inhaler, for the treatment of COPD. According to the manufacturer, Combivent is indicated for use in patients with COPD who use an aerosol bronchodilator and continue to have evidence of bronchospasm and who require a second bronchodilator to dilate constricted airways. For a list of indications, contraindications, and side effects, circle Reader Service Number 164.



INFANT APNEA-RESPIRATION MONITOR. Electronic Monitors International announces the model RE-134 C, a small, lightweight apnea-respiration monitor

with automatic sensitivity control, respiratory signal indicator, and an audial-visual apnea alarm with a 20-second preset delay. The manufacturer claims this system is an inexpensive way to monitor apnea respiration in the hospital nursery without using electrodes or lead wires. In addition, a small respiration sensor placed beneath the patient's mattress detects the slightest respiratory signal using patented technology developed by NASA. Model RE-134 D offers a liquid-crystal display digital readout of respiration rate in breaths/minute. Circle Reader Service Number 165.



ELECTROENCEPHALOGRAM (EEG) SYSTEM. Medilog® Vision, the fourth generation of ambulatory EEG systems from Oxford Instruments Medical Systems Division, features digital replay of EEG, automatic staging and analysis of sleep, synchronized video for long-term monitoring, and digital EEG capabilities linked through a reader station. According to a company press release, patient data, via 16 channels for EEG and 1 dedicated channel for electrocardiogram recordings, can be collected for up to 24 hours on Oxford's small, lightweight Medilog MR95 digital recorder. In addition, the Medilog Vision system rapidly replays the recorded data at up to 100x real time and can display information in a wide variety of montage formats. For more information, circle Reader Service Number 166.

Policy and Procedure Manual

Save time and money by making your department more efficient with the *Policy and Procedure Manual.* Its 130 pages of policies and procedures cover the aspects of administrative and clinical respiratory care for both adult and pediatric practice. Sections on administrative policies, therapeutics, clinical monitoring, and mechanical ventilation. Standardized formats include objectives, indications, equipment, policies, contraindications, troubleshooting, procedures, hazards, and assessment of effectiveness.

Item BK6 $60 ($70 nonmembers)

Uniform Reporting Manual

The *Uniform Reporting Manual* provides you with nationally recognized standards for documenting workload units and time standards. Includes patient assessment activities and covers bronchial hygiene, supplemental oxygen, airway care, diagnostic tests, and cardiovascular diagnostics. In addition, there are chapters on "Clinical Activities Without Time Standard" and "Management Support Activities." *By the AARC. 1990, updated 1993. Binder, 165 pages.*

Item BK1 $65 ($85 nonmembers)

Respiratory Home Care Equipment

Respiratory Home Care Equipment exclusively covers home care equipment with practical applications and charts on oxygen concentrators, liquid units, air compressors, and home care ventilators. An invaluable book for the home care practitioner and equipment technician. Details home care devices, cleaning, disinfecting, and monitoring procedures to minimize infection. Includes guides for educating staff on equipment, therapy, patient assessment, and safety. Features procedures for gas administration and monitoring devices, humidifiers and nebulizers, artificial airways and resuscitators, respirators, and ventilators. *By Steven P. McPherson. Hardcover, 192 pages, 141 illustrations, 6 tables. 1988.*

Item BK7 $9 ($12 nonmembers)

A Study of Chronic Ventilator Patients in the Hospital

Chronic ventilator-dependent patients are costing American hospitals more than $9 million per day according to this Gallup study conducted for the AARC. This important study provides information on patients who depend on life-support systems; why, how, and where they are being treated; and the cost of treatment. *47 pages, 9 tables, 12 figures.*

Item BK20 $25 ($50 nonmembers)

The AARC Human Resources Survey: A Study of Respiratory Care Human Resourceslin Hospitals

Covers a wide range of human resource issues, including compensation, numbers of full-time equivalents, job vacancy rates, education, credentialing, and licensure. Even includes information on age, sex, and years of experience. Includes comprehensive summary, position profiles, salaries, education, experience, credentials, and regional demographics. Vacancies are inventoried. *68 pages, 66 tables.*

Item BK12 $35 ($50 nonmembers)

A Study of Respiratory Care Practice

This study examines the practice of respiratory care in today's health care environment and how hospital reorganization is affecting the profession. Includes chapters on medical direction, current respiratory care services, nontraditional services, respiratory care protocols, and hospital service reorganization. *38 pages, 34 tables, 15 charts.*

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Extracorporeal Life Support Organization
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U.S. Department of Health and Human Services, Health Advice
http://www.healthfinder.gov

Food and Drug Administration
http://www.fda.gov

National Asthma Education Prevention Program Guidelines, 2nd Edition
http://www.nhlbi.nih.gov/nhlbi/lung/asthma/prof/asthgdln.htm

The Lambda Beta Society
The National Honor Society for the Profession of Respiratory Care

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The National Board for Respiratory Care—1998 Examination Dates and Fees

Examination	Examination Date	Examination Fee
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	July 11, 1998 Application Deadline: May 1, 1998	100 (new applicant)* 60 (reapplicant)*
	November 14, 1998 Application Deadline: September 1, 1998	100 (new applicant)* 60 (reapplicant)*
RRT Examination	June 6, 1998 Application Deadline: February 1, 1998	120 Written only (new applicant) 80 Written only (reapplicant)
		130 CSE only (all applicants)
	December 5, 1998 Application Deadline: August 1, 1998	250 Both (new applicant) 210 Both (reapplicant)
CPFT Examination	June 6, 1998 Application Deadline: April 1, 1998	130 (new applicant) 100 (reapplicant)
RPFT Examination	December 5, 1998 Application Deadline. September 1, 1997	180 (new applicant) 150 (reapplicant)
Perinatal/Pediatric Respiratory Care Specialty Examination	March 14, 1998 Application Deadline: November 1, 1998	180 (new applicant) 150 (reapplicant)

*In 1999, this examination fee will increase by $20.

For information about other services or fees, write to the National Board for Respiratory Care, 8310 Nieman Road, Lenexa KS 66214, or call (913) 599-4200, FAX (913) 541-0156,or e-mail: nbrc-info@nbrc.org

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185	186	187	188	189	190	191	192	193	194	195	196	197	198	199

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3 ❑ Critical Care
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7. ❑ Management
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9. ❑ Rehabilitation
10. ❑ Education

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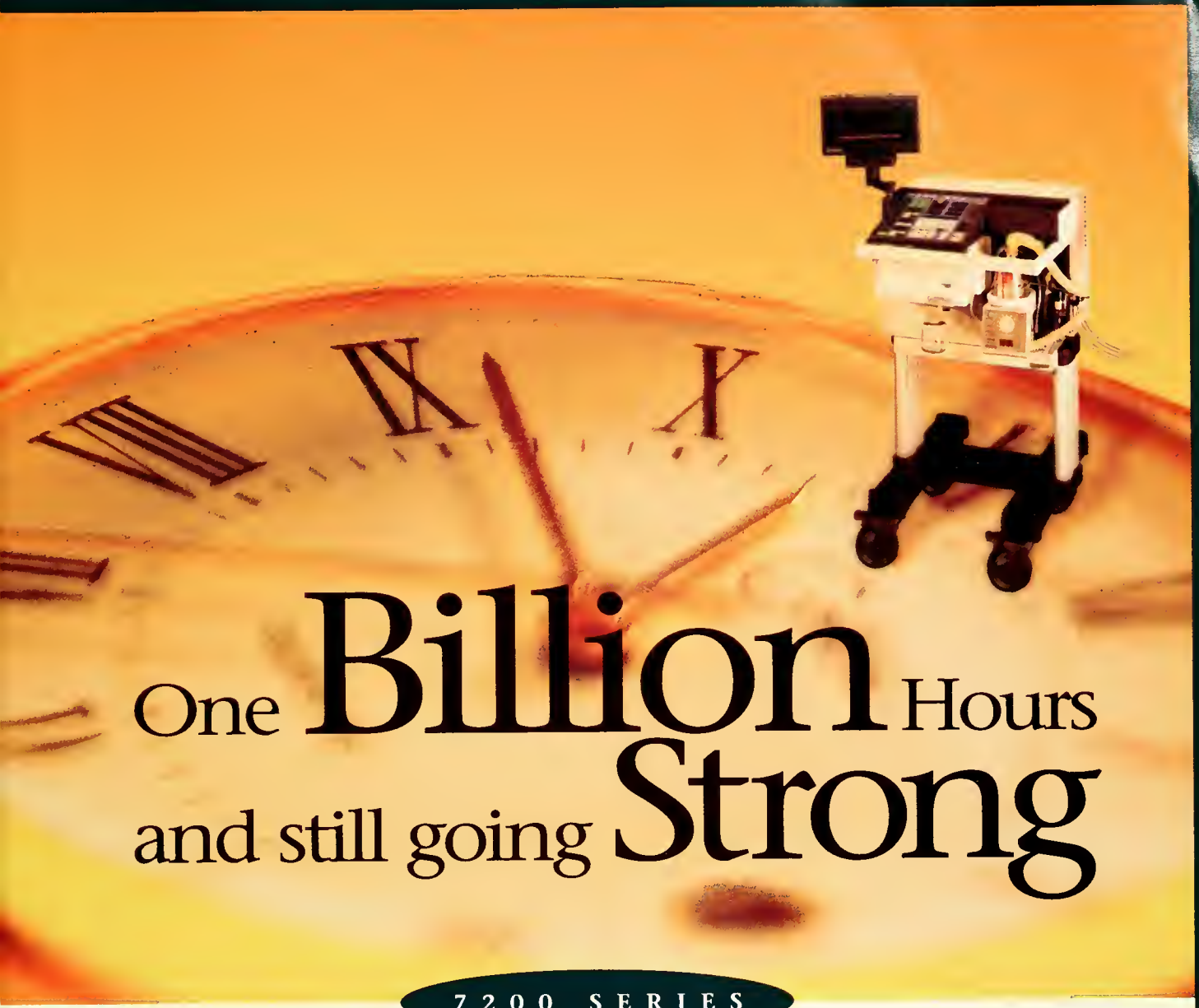

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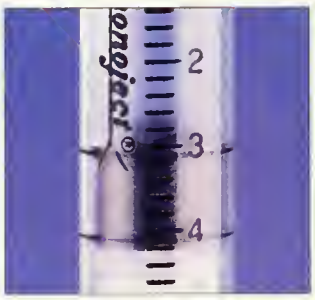

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